LIX

SCLEROMA IN GUATEMALA

WITH A STUDY OF THE DISEASE BASED ON THE
EXPERIENCE OF 108 CASES

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Guatemala is a mountainous, tropical country of about 43,000 square miles, with cities, towns and villages located at an altitude ranging from sea level (Atlantic and Pacific) to 8,000 and even 10,000 feet. A good percentage of the population is composed of pure Indians who still live a kind of life as primitive and wild as that of their ancestors of 500 years ago. The rest of the people are "mestizos," varying from the almost pure Indian to the almost pure Spanish type.

Historians and medical investigators have not solved many problems in relation with several diseases that we see now in the Western Hemisphere: syphilis, leprosy, yaws, etc., have been the subject of interminable debates, in an attempt to establish definitely whether they existed in the New World prior to its discovery, or whether they were brought by the conquerors, together with their religion, their language, their way of life, their civilization. As long as this work deals with scleroma in Guatemala, the first questions that come to our mind are: Did this disease exist in the American

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continent before 1492? Was it brought by the Spaniards? Or was it carried to the Americas later by other immigrants from the Old World? We wish we were able to answer these questions; but the problem is not a simple one and still remains unsolved.

HISTORY

The Mayan civilization is the oldest of the Western World, and the oldest Mayan city can be admired in the ruins of Tikal, in Guatemala. The ruins of Uaxactún, Quiriguá and others, are also indestructible vestiges of the Guatemalan part of the ancient Mayan Empire. As far as we know, nothing has been found among the unburied relics of the Mayans which would make us suspect that a disease like scleroma, in any of its forms, was prevalent or even existed in that extinguished race.

The same can be said about the descendants of the Mayans, the Indians that the conquerors found in the Western World. Although the sacred books of the Indians are full of observations related to the science of medicine and some diseases are thoroughly described, there is nothing to make us accept the idea that scleroma was an indigenous affection. Based on information gathered through exhaustive studies of Central and South American history and archeology, recent investigators have proved, for example, that syphilis was prevalent among the natives of the Western Hemisphere before 1492, and it has also been proved that this disease existed in the Old World long before the discovery of America. In regard to scleroma, we have not been able to find anything to conclude that our pre-Columbus Indians ever saw this disease. Moreover, our statistics show that pure Indians are affected by scleroma in a much lower proportion than those who have even a small percentage of European blood.

It is impossible to know when scleroma appeared in the American continent for the first time, but we have every reason to state that it attacked the native population long before von Hebra considered scleroma an individual pathological condition. When our physicians of the nineteenth or even the eighteenth centuries spoke of "nasal leprosy," "hard nose," "rotten nose," "fetid nose," "exuberant syphilis of the nose," "chronic catarrhal croup," etc., we know that some of those names must have been applied, at least sometimes, to cases of rhinoscleroma or laryngoscleroma. We do not know how or when the disease was brought to Central America, but we do know that soon after von Hebra's publication[86] in 1870, investigators in El Salvador and Guatemala identified the disease and discovered that scleroma could be easily found and diagnosed in these two countries.

MAP OF GUATEMALA
Showing the geographical
distribution of 108 cases
of Scleroma

Fig. 1.

In 1876 Mikulicz[59] published his studies on microscopic histology of scleroma and spoke for the first time of the "swollen cells," which since then bear his name. In the same year Gerhardt gave the first comprehensive description of laryngeal scleroma. In 1882 von Frisch[85] discovered, in the nose of patients suffering from rhinoscleroma, a bacillus which he thought was the causal agent of the disease.

In Central America several investigators soon were busy with the problem of scleroma. In 1883 Guevara,[37] of El Salvador, wrote a thesis on "Scrofulous Lupus," a name that had been suggested by Rösser; Guevara reported 11 cases of scleroma and confirmed in El Salvador most of the findings of the European authors.

In 1898 the work of Alvarez,[7] of El Salvador, was published, summarizing the results of many years of clinical and bacteriological research; he reported 22 cases of scleroma. But the reason why
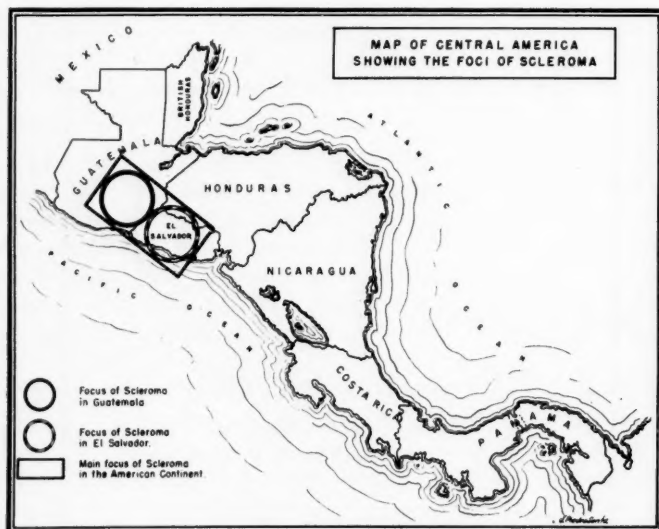
Fig. 2.

Alvarez deserves a prominent place in the history of scleroma is his discovery, in the leaves of a leguminous plant, "jiquilete" (*Indigofera tinctoria*), of a bacillus morphologically similar to von Frisch's bacillus. Scleroma being prevalent, according to Alvarez,[8] among aniline dye workers, and the dye (indigo) being obtained from the named plant, he concluded that his bacillus (*Bacillus indigogenous*) and *B. rhinoscleromatis* of von Frisch were the same organism. His bacteriological findings were confirmed by Professor Lafar, of Vienna, and by other workers; but his theory that both micro-organisms were identical and that *B. indigogenous* was the pathogenic agent of scleroma in El Salvador and Guatemala was not backed by other investigators who were interested in his work.

Gutiérrez,[39] in Guatemala, after years of study and research, wrote, in 1899, a magnificent monograph on scleroma, and reported 21 cases. His work included an exhaustive bacteriological study of the disease, animal inoculations—which were practically unsuccessful—a review of the literature and a critical analysis of the different treatments in use at that time. Reading the work of Gutiérrez we find very interesting information. For instance, he tells us that

Bulkley was the first one to report a case in the United States. However, in the modern American bibliography we find that the first case was reported by J. T. Jackson, in 1893. We cannot help asking ourselves: Who was Bulkley? Why is his name not even mentioned in the literature?

In 1901 Sánchez,[73] of Guatemala, wrote a thesis on the surgical treatment of rhinoscleroma, and reported 32 cases. Before the Fifth Pan American Medical Congress, in 1908, Wunderlich, of Guatemala, presented three cases of scleroma cured with x-rays. Quiñonez,[70] of El Salvador, reported, at the same Congress, ten other cures with x-ray therapy. In 1910 Wunderlich[92] reported another series of 14 patients with whom roentgen therapy was used.

Grajeda,[36] in 1925, presented before the Guatemala University School of Medicine a work on the use of radium in the treatment of scleroma. He reported 29 cases. Reyes,[71] of El Salvador, published in 1946 some observations on the subject of scleroma, based on the study of 200 cases.

GEOGRAPHY

At first it was thought that scleroma existed only in a limited region of eastern Europe. Then, as the disease became better known to physicians, cases were reported from everywhere. However, we have to admit that there are, scattered around the world, several real foci of scleroma: southwestern Russia, Poland, Hungary, the Balkan peninsula, Italy, Egypt, the Sunda Islands, Mexico, El Salvador, Guatemala, etc. If we study each one of the recognized foci of scleroma we find that even in each country or locality there is a more circumscribed and limited region, which constitutes the actual focus, the nucleus of the area: the Ukraine in Russia, Galicia in Poland, Pasoemah and Minahassa in the Sunda Islands, the central states in Mexico, the southeastern states in Guatemala, etc.

This work is based on the study of 108 cases of scleroma seen at the Guatemala City General Hospital during the last five years. These cases represent at least 90% of all the cases of scleroma observed in the whole country, because the above named hospital is the only one where x-ray therapy and radium therapy are available. Patients who came from neighboring countries, and also those in which sufficient information was not obtained are not included in our series.

Care was taken, in each one of our cases, to find out as precisely as possible where the patient was living at the very beginning of

the disease. This was not difficult because Guatemalan peasants are rather sedentary people. Figure 1 shows a map of the whole country, where each one of the 108 cases is represented by a black dot. The first thing that strikes us when we look at the map is that the northern and western parts of the country are comparatively free of scleroma, and that in the central and southeastern states the disease is prevalent. If we join this finding to the fact that hundreds of cases have been reported in the neighboring country of El Salvador we have to draw two conclusions (Fig. 2): (a) the main focus of scleroma in the American continent covers El Salvador, with an extension of 13,000 square miles, and an area of approximately the same size in southeastern Guatemala; (b) there must be a scleroma region in southwestern Honduras, in the immediate vicinity of Guatemala and El Salvador, because the geographic, climatic, ethnic and economic conditions of that area make it extraordinarily adequate to complement the Guatemala-El Salvador focus. We have made an inquiry among Public Health officials in the whole country, and the reports coincide with our statistics: scleroma is practically unknown in the northern and western sections of Guatemala.

There is scleroma in Mexico, as well as in Cuba and Colombia, and there have been cases reported in every country of the Americas; but we do not know of another area so clearly limited as the Guatemala-El Salvador scleroma region. About Brazil, there has been a mistake: some writers state that scleroma is endemic in that country because they have taken the city of Salvador (in Brazil) for the republic of El Salvador (in Central America). However, some cases have been reported in Brazil (26 up to 1947), in the same sporadic fashion as in any other part of the world.

RACIAL DISTRIBUTION OF 108 CASES

INDIANS	WHITES	MESTIZOS	NEGROES	OTHER RACES	TOTAL
7	0	101	0	0	108

Studying our series we find that in Guatemala the disease is more common above 2,000 feet and that it is almost unknown in the lowlands. Seventy per cent of our cases came from towns and villages above 4,000 feet from sea level.

INCIDENCE

It is universally admitted that scleroma is a disease of dirty individuals; very well known is the eloquent expression, "Disease of the great unwashed." However, we have had the opportunity of

seeing scleroma in particularly clean persons. Our Indians, especial-
ly those of the highlands, lead a filthy and miserable life, and it is
interesting to note that in our series less than 7% of the cases are
pure Indians; the rest are "mestizos" with cleaner habits and more
"civilized" living conditions.

Scleroma is a disease rarely observed in children. The youngest
patient in our statistics is a girl 8 years old; the oldest one is a man
of 68. The following chart summarizes the age incidence in our
cases:

AGE GROUP	NUMBER OF PATIENTS	PERCENTAGE
0-10	1	0.93
11-20	21	19.44
21-30	39	36.11
31-40	28	25.93
41-50	10	9.26
51-60	5	4.63
61-70	4	3.70

We have not found a single author who does not agree with
the known statement that scleroma attacks females in a higher
proportion than males. Belinoff's report,[11] for instance, shows an
incidence of 58.5% for females, and 41.5% for males. In Guatemala,
scleroma apparently does not follow the rule. According to Gutié-
rrez,[39] in the Guatemala City General Hospital there were 18 cases
from 1887 to 1897, 13 males and 5 females; Gutiérrez himself re-
ported another series of 13 males and 8 females. The cases re-
ported by Sánchez[73] in 1901 were 19 males and 13 females. Grajeda's
cases,[36] however, were 12 males and 17 females. Our cases are as
follows:

MALES	65	60%
FEMALES	43	40%

About the occupation of the patients, most of our cases are
destitute peasants; a few live in the slums of towns or cities. A
great majority of the men are rural laborers, but there are two
shoemakers, one carpenter, four masons, two blacksmiths, three
musicians. Most of the women are rural home workers.

CLINICAL PATHOLOGY

Scleroma is a chronic, possibly specific, granuloma of deceiv-
ingly benign symptoms at the beginning, and characterized by a
slow and treacherous progress; it fully deserves to be called insidious
disease.

For didactic reasons, when describing scleroma (rhinoscleroma) most writers divide its course in three stages or periods. We shall follow the same method, but it has to be borne in mind that those stages do not exist as such, and that the course of the disease does not follow any rule or any pattern.

First Period. Some authors call it "rhinitis stage"; others, "catarrhal stage"; and still others, "atrophic period." The words "stage" or "period" are used in an indifferent way. Very few have really seen the beginning of scleroma. It may start with the symptoms of atrophic rhinitis or with those of hyperplastic rhinitis. In our experience the main complaint at the beginning is dryness of the nose, with sensation of obstruction of the nostrils. The process is, in the great majority of cases, bilateral and symmetrical; there is no pain or fever; as a rule, there is secretion—mucous, purulent or crusty—because there is always a rhinitis. Anterior rhinoscopy shows a narrowing of the airway, sometimes very slight. The nasal membrane is usually congested and gives the impression of being thickened; it bleeds easily and sometimes has, particularly in the vestibule, small fissures covered with minute, brown scabs. Posterior rhinoscopy does not show anything during the first stage, except, undoubtedly, in cases of primary scleroma of the nasopharynx, which we have never seen. Examination with the nasopharyngoscope confirms the findings of anterior and posterior rhinoscopy. If we were allowed to subdivide the first period into two others we might say, in this description of the usual case, that rhinoscleroma first resembles chronic diphtheria of the nose, and later it shows a picture more similar to atrophic rhinitis. There is no odor during the first stage, although it is almost the rule to see small, thin, moist crusts. X-ray pictures taken during the rhinitis stage usually fail to show anything abnormal; occasionally slight thickening of the lining of the sinuses or relative obstruction of the nose are reported. There is no visible deformity of the nose. The lymphatic glands are not affected. The general condition of the patient is not altered by the disease.

Second Period. Called "infiltrating period," "granulation stage," or "granulomatous stage," the second period is characterized by increased obstruction of the nose and visible external deformity, due to invasion of the soft structures by granulomatous tissue. The mucous membrane of the nose gets thicker and harder, especially on the septum and on the floor of the nasal cavity; the tip of the nose gets hard, the columella loses its mobility and the upper lip starts to show some deformity due to subcutaneous hard swelling. When the lesion advances backward into the nasal cavity,
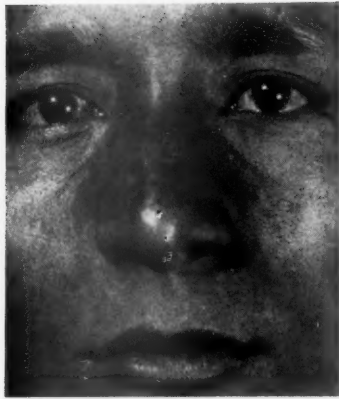
Fig. 3.—Mild case of rhinoscleroma of two years' duration. There is
a prominence on the right side of the nose and asymmetry of the nostrils.

it may reach the soft palate and the nasopharynx; if the soft palate
is invaded it becomes thick and hard. Anterior rhinoscopy reveals
the narrowing of the airway, sometimes to the extent of making
breathing through the nose extremely difficult; there is thick, yel-
low, greenish or brown discharge from the nose, and also a char-
acteristic, foul odor. Posterior rhinoscopy shows how far the disease
has spread towards the pharynx; the thickness of the mucous mem-
brane, the invasion of the soft palate and the extension to the eusta-
chian tubes can be easily appreciated through posterior rhinoscopy.
The nasopharyngoscope, when the obstruction does not prevent its
use, is particularly helpful for the inspection of the eustachian tubes.
As a rule, the scleromatous lesions have no tendency to show ulcera-
tions, unless they are traumatized. As long as the disease advances
the signs and symptoms become more pronounced, and the spread
of the scleromatous invasion may cause great functional disorders
and ghastly deformities. The cheeks, the whole upper lip, the gums,
the anterior and posterior pillars, the pharynx, may be invaded by
extension of the disease. Sometimes scleroma, during the granu-
lomatous period, may be exuberant and take the appearance of gran-
ulation tissue; in this case epistaxis is usually a troublesome symptom.

In most cases scleroma causes no pain, except the pain or head-
ache that may be due to the obstruction of the nose. Anesthesia
of different areas may be observed as the disease advances; a fre-

quently observed symptom, even during the first period, is the paresthesia or anesthesia of the soft palate. X-ray examination during this second stage shows marked thickening of the lining of the sinuses and, not infrequently, retention of fluid; in the very serious cases the x-ray pictures present, besides the obstruction of the nasal cavity, the displacement of the hard structures.

The patient usually complains of a variety of symptoms in relation with the advancement of the disease and the obstruction of the nasal cavity: frontal headache, pain over the region of the maxillary sinuses, earache, anosmia or cacosmia, epiphora, tinnitus, impairment of hearing, postnasal discharge, dryness of the throat, sore throat, etc. It is during this second stage that the larynx and the trachea may be invaded by the process, presenting the series of symptoms and signs that are described below.

The general condition of the patient is affected more by the secondary infection and the functional disturbances than by the scleromatous process itself. The lymphatic glands are still spared by the disease.

Third Period. "Nodular period," "stage of fibrosis," this phase is reached when the infiltration becomes harder, due to the substitution of the granulomatous tissue by firm, contracting scar tissue. As a rule, the disease goes into the nodular stage after several years of ever increasing granulomatous infiltration. It should be clearly stated that the nodular stage means that the disease has reached its peak, especially as far as external deformity is concerned, and that from then on a relative regression can be expected. There is a change of the granulomatous infiltration into sclerotic and fibrotic tissue. The stage is called "nodular" because the process affects the various regions in an irregular form, causing the formation of nodules. In the nasal cavity the sclerosis and the formation of scars make the membrane dry and harder; the retraction of the connective tissue increases the obstruction of the nares, and sometimes the scar tissue completely closes the airway. The nodules are small, localized at the beginning in the anterior part of the nasal cavity, particularly on the septum and the floor of the nose. Later they spread in all directions and also coalesce to form larger nodular masses. It is during this period that the soft palate, even when slightly involved, suffers an upward retraction, as if pulled from the nose by symmetrically attached strings; frequently only the tip of the uvula is visible, occasionally the whole structure disappears into the nasopharynx. If the pillars were involved during the second stage, they become fibrotic, and adhere to the neighboring regions, pulling the base of the tongue upwards and also narrowing the
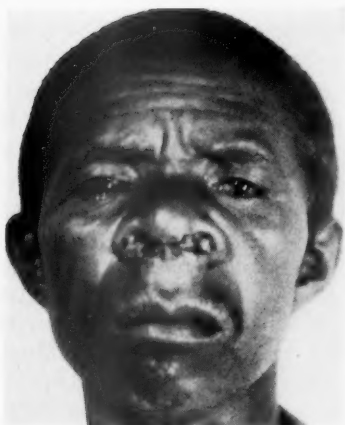
Fig. 4.—Advanced case with almost complete obstruction of the nares.

Fig. 5.—Same case as Fig. 4.

communication between the oropharynx and the nasopharynx. Look-ing through the mouth of a patient who has had involvement of the soft palate and the pillars, we can see, during the nodular period, a classical picture of scleroma that might be called "gothic palate."

During the infiltration period the deformity shows its maxi-mum extension; but it is during the third period that this deformity gets irregular, "nodular," particularly on the outside; the whole nose acquires a stone-like consistency, and firm, small, round masses (nodules) are visible, covered only by a thin, shiny skin. The sites of election of these masses are the columella, the upper lip and the alae of the nose; nevertheless, they may be seen in the neigh-boring areas, according to the extension of the lesions. On the upper lip the nodules are usually bilateral and symmetrical, making a prominence just beneath the nostrils; it is not rare to see the colu-mellar nodule extend downwards under the filtrum and in this way contribute to the deformity of the upper lip. X-ray examination during the third period shows the same picture as during the second one. In some cases, however, a process of bony sclerosis can be de-tected, very similar to the sclerosis observed in the temporal bone in cases of chronic infection of the ears; we feel that it is rather a re-action to the secondary infection than to the scleromatous invasion.

The constitutional condition of the patient remains stationary; but the secondary infection and the defective respiration are respon-sible for the intercurrent diseases that may attack the patient. Bronchitis, bronchiectasis, tuberculosis, pneumonia, bronchopneu-monia, etc., are frequently seen in patients with rhinoscleroma. The life expectancy in advanced cases of scleroma is greatly diminished, and death comes—frequently as a blessing—as the result of an intercurrent disease.

Rhinoscleroma neither follows any rules nor adapts itself to any pattern; consequently, no description of this disease can be complete. All cases are different: some are very benign and limited in extension, others show marked invasion by the disease, with tre-mendous deformities; and between these two extreme cases we find the most varied scale of possibilities. However, there is always some-thing common to every case of rhinoscleroma; unfortunately, that "something" cannot be described with words.

Laryngoscleroma and Tracheoscleroma. Primary scleroma of the larynx or trachea is a very rare event. There is only one case in our series (laryngoscleroma); the patient is a 14-year-old girl who, three years after the beginning of the laryngeal symptoms, started to show signs of invasion of the nasal mucosa, in a very mild way.

Fig. 6.—Rhinoscleroma, nodular stage.

Fig. 7.—Same case as Fig. 6.

Secondary scleroma of the larynx, on the contrary, is a common complication of rhinoscleroma. In our series we have 22 cases, or 20%. Tracheoscleroma is not so frequent, and as a rule, it is an extension of the laryngeal lesion. We found it five times, 4.6%, never without involvement of the larynx.

It is during the infiltration stage of rhinoscleroma that the disease usually attacks the larynx and the trachea; we may say that if the disease has reached the nodular period the patient has a good chance of escaping the danger of laryngeal and tracheal complications. It has been stated that the laryngeal lesion is always subglottic. With this we do not agree; frequently the process starts below the vocal cords, no doubt, but we have seen scleroma start in the false cords, in the interarytenoid region, in the aryepiglottic fold, and even stay for years localized in the epiglottis, without extension to any other part of the larynx.

The symptoms vary with the site of the lesion. Subglottic scleroma is the one which makes the condition more uncomfortable and dangerous; in a great majority of cases, no matter where laryngoscleroma has started, the subglottic region sooner or later gets involved. The symptomatology is that of chronic laryngitis; the patient complains of dryness "down in the throat," has to "clear" his larynx, his voice is changed. If the patient is examined at an early stage it can be seen that the beginning is a thickening of the mucous membrane, with visible dryness of the area, as if the epithelium were losing its glands. When the subglottic region is affected the thickness of the mucosa is clearly seen as a relative narrowing of the airway just below the vocal cords. Later on, the region is observed covered with sticky crusts, and the infiltration spreads to the vicinity. When the vocal cords are involved—which is the rule in advanced cases—they get red, thick, crusty and partially fixed. The interarytenoid region is apt to show edema, sometimes so marked that it makes it impossible to visualize the glottis by indirect laryngoscopy. The patient's complaints are in accordance with the progress of the disease: hoarseness and coughing are usually present; scant, thick, crusty, sometimes sanguineous expectoration which annoys the patient, is also observed. As a rule, there is no pain, but some difficulty on swallowing can be complained of. The dangerous and most distressing symptom is dyspnea, which is the natural consequence of the advancement of the disease: too frequently a tracheotomy has to be performed in order to save the patient's life.

Tracheoscleroma is the extension of laryngoscleroma in a downward direction. Usually it does not go beyond the first five tracheal rings, although exceptionally it may affect the rest of the trachea
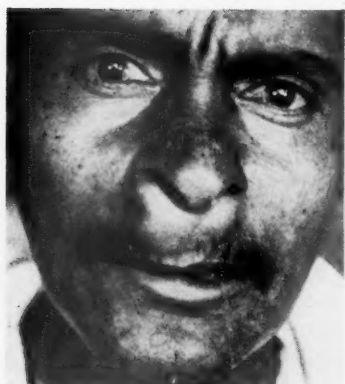
Fig. 8.—The disease has invaded the upper lip where several nodules have coalesced to form a large scleromatous mass.

Fig. 9.—Same case as Fig. 8.

and the main and even the secondary bronchi. Through the bronchoscope, or on the operating table, the lesions look like soft, vascular masses resembling granulation tissue; they are very movable and frequently interfere with respiration in a valve-like manner. Occasionally the tracheal lesion consists of a dry and hard, concentric narrowing of the lumen of the trachea. Tracheoscleroma being an extension of laryngoscleroma, it adds to the latter's symptoms those related to the obstruction of the trachea.

MACROSCOPIC PATHOLOGY

Scleroma is characterized by a granulomatous infiltration of the soft tissues and by the formation of nodules or tubercles, which are present during the late stages of the disease. Cartilage and bone are always spared, but we have observed a marked softening of both structures; bone may be affected by a process of sclerosis during the late stage. When the skin or mucous membranes become ulcerated, a portal of entry for secondary infection is open; this increases the peculiar odor of scleroma and contributes to the spread of the disease.

If a piece of tissue is taken for biopsy, or a minor operation performed, it can be appreciated that the tissue is very soft, giving the impression of going through a process of "disintegration;" during the nodular stage, on the contrary, the tissues are firm, sometimes stone-like in consistency. It is very frequent to find firm, scarred areas about the nostrils, and soft spots closer to the nasopharynx, where the tissues have not undergone the changes that characterize the nodular stage. Cartilage and bone can be pushed by the scleromatous invasion in such a form that the normal anatomical relations may be fundamentally changed; the bones that first yield to the attack are those of the lateral wall of the nasal cavity, the nasal bones and the ascending process of the superior maxilla. We have seen, at the autopsy table, the inferior turbinate pushed into the antrum and this cavity completely disappear as a consequence of rhinoscleroma of unusual severity; in the same case scleromatous tissue was found in the eustachian tubes and in the left middle ear. Chronic, bilateral, middle ear infection is present in a good percentage of the cases of rhinoscleroma. In the larynx the process usually leads to concentric obstruction; we have not seen this kind of obstruction in the trachea or bronchi, where it is caused mainly by exuberant scleromatous tissue. We have not observed concentric obstruction below the larynx.

The lymphatic glands are not affected by scleroma; when they become enlarged it is as a result of the secondary infection.

Fig. 10.—Exceptional case of uncontrolled scleromatous infiltration.

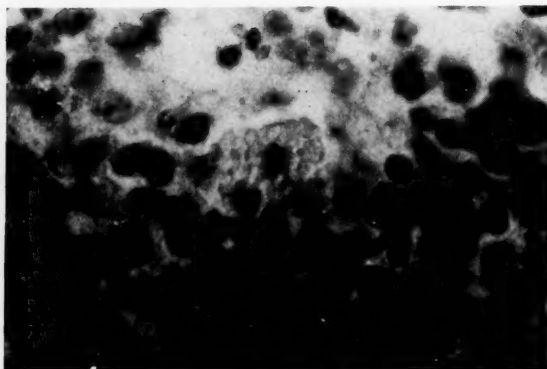Fig. 11.—Same patient as shown in Fig. 10.

Fig. 12.—Section of scleromatous tissue. Large Mikulicz cell show-
ing the characteristic "foamy" cytoplasm.

MICROSCOPIC PATHOLOGY

Very little has been added since the magnificent histologic stud-
ies of the early investigators of scleroma. Gerber, in 1872, gave a
beautiful description of the microscopic picture of this disease, being
followed, to mention the outstanding works, by Mikulicz[59] (1876),
von Frisch[85] (1882) and Unna[84] (1903).

The process is a granuloma. But besides the picture of granu-
loma there are the microscopic findings of chronic inflammation
and three almost diagnostic elements: the Mikulicz cell, the Rus-
sell body and the von Frisch's bacillus.

The epithelium may show atrophy or hyperplasia as a result
of the underlying process. Polymorphonuclear leukocytes, small
lymphocytes, eosinophilic leukocytes and plasma cells are always
found. During the first stage of the disease a microscopic diagnosis
cannot be made.

During the infiltration period the "swollen cells" (geblähte
Zellen) of Mikulicz make their appearance. Also called "foam,"
"lace" and "bubble" cells, they are large cells (20-50 microns in
diameter), with the nucleus surrounded by granular, foamy cyto-
plasm. The Russell bodies, also called hyaline bodies or Unna cells,
are uniformly translucent cells which take easily the acid stains;
they are 10-40 microns in diameter and may or may not show a
nucleus; when they do, it is usually at the periphery of the cell;
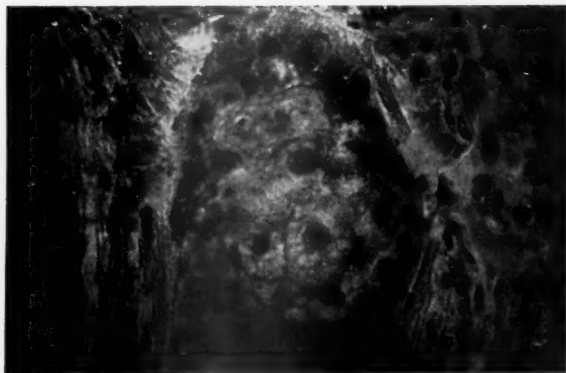occasionally, the hyaline body seems to be caught during the process

Fig. 13.—Group of Mikulicz cells, probably within a lymphatic vessel.

Fig. 14.—Mikulicz cell in the center of the picture showing intracellular *Klebsiella* organisms.

of extruding the nucleus. Both the Mikulicz cells and the hyaline bodies are believed to be degenerated leukocytes. The bacillus of von Frisch (*Klebsiella rhinoscleromatis*) is easily detected in the sections of scleromatous tissue; it may be seen within the Mikulicz cells or in the lymph spaces.

Nothing is pathognomonic in the histology of scleroma. Mikulicz cells are also found in leprosy, bubonic plague and other diseases. Russell bodies are found in specific and nonspecific granulomas and in some malignant neoplasms. But if the microscopic examination of a piece of tissue shows a granuloma, with Mikulicz cells, hyaline bodies and von Frisch's bacilli, a certain diagnosis of scleroma can be made.

BACTERIOLOGY

Klebsiella rhinoscleromatis, or von Frisch's bacillus, is a short, encapsulated, gram-negative diplobacillus, with round ends, 2-6 microns long and 0.5-1 micron wide. When von Frisch[85] discovered the micro-organism and succeeded in cultivating it, he thought it was the causal agent of scleroma. Nowadays there are authors who still sustain von Frisch's theory; there are others, however, who consider the presence of *K. rhinoscleromatis* as secondary to the obstruction of the nose and to the degeneration of the tissues. The fact that the bacilli are present in other diseases and even in healthy persons comes to support the case against von Frisch's original idea. According to Watkins,[88] Babès found von Frisch's bacilli in 50% of the cases of chronic inflammation of the nasal mucosa, and Neumann found it in 20% of normal persons.

K. rhinoscleromatis is similar to *K. pneumonia* (Friedländer's bacillus) and to *B. mucosus capsulatus*. Some bacteriologists think all of them are only one micro-organism and know it as *Klebsiella* type C.

In Guatemala, Gutiérrez,[39] in 1899, made a beautiful bacteriological study of scleroma and successfully cultivated the bacillus in most of the usual laboratory media. He did not succeed, however, in reproducing the scleromatous lesions by inoculation of animals. It can be said that such attempts at reproducing the disease by injection of *K. rhinoscleromatis* have always failed. The organism can be easily obtained and cultivated from almost every case of scleroma. In our certainly limited experience, it grows best in human blood agar, where it forms "mucoid" colonies; these colonies, when fully grown, may reach the size of 10 mm in diameter, become white and show the classical appearance of paraffin drops.

Gram-negative *K. rhinoscleromatis* takes the ordinary laboratory dyes, and its capsule can be demonstrated by any of the capsule staining techniques.
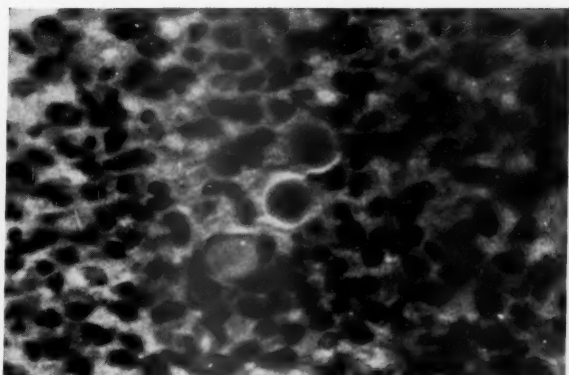
Fig. 15.—Hyaline bodies (three in the center of the picture).
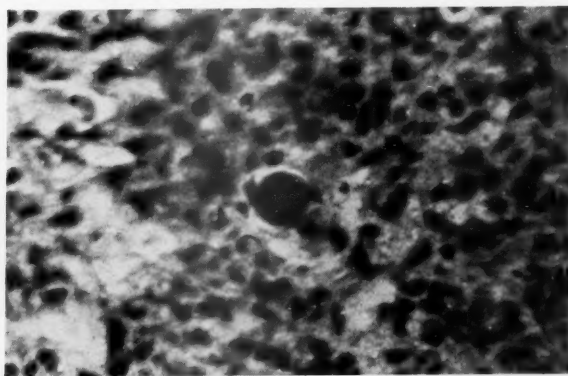

Fig. 16.—Hyaline body deeply stained.

Cultures from nasal secretions or from diseased tissue in cases of scleroma also show what might be expected: (a) micro-organisms that are ordinarily found in a healthy nose; (b) all the germs that may cause secondary infection; (c) some saprophytic bacteria.

ETIOLOGY

Is the bacillus of von Frisch the causal agent of scleroma? A good number of bacteriologists sustain that von Frisch's bacillus and Friedländer's bacillus cannot be differentiated from each other. Many have obtained *K. rhinoscleromatis* from the nasal cavity of patients affected by diseases other than scleroma, and even from the nose of healthy persons. Inoculations of the bacteria into the body of animals or human beings have failed to reproduce the scleromatous lesions. Based on those facts a school of thought sustains that the etiology of scleroma is a problem still unsolved. Other investigators defend the original hypothesis of von Frisch. We belong to the first ones.

Cunning and Guerry[22] suggested, in 1942, the idea that scleroma "might be caused by a virus, or perhaps a virus and the von Frisch bacillus acting symbiotically." So far, no light has come from this angle into the obscure problem of the etiology of scleroma. Reyes,[71] of El Salvador, is working on the same line, but, as far as we know, his studies have not been reported.

Does heredity play a part in the etiology of scleroma? Apparently it does not. Pjayak has proved in an exhaustive investigation that heredity should not be considered as a factor of importance in the etiology of scleroma. In our experience we have never found a single case in which heredity could be blamed.

Is scleroma a contagious disease? We think it is, but in a mild degree. The fact that there are regions where the condition is definitely prevalent and the numerous cases observed in members of the same family, or in persons living in the same household, force us to believe that contagion plays a part in the etiology of scleroma. In our series, cases 46 and 102 are sisters, and cases 51 and 106 are brothers. In the records of the Guatemala City General Hospital, Department of Radiology, we found four other cases—not included in our statistics—of two brothers and two sisters of the same family, who, affected by rhinoscleroma, received roentgen therapy from 1935 to 1937. Very interesting is the report of Lasagna[50] of an Italian family in which 15 persons were affected by scleroma. Of greater interest is the report of Hara et al.[41] of seven cases of scleroma in one family in the United States, a country where the disease is rarely seen.
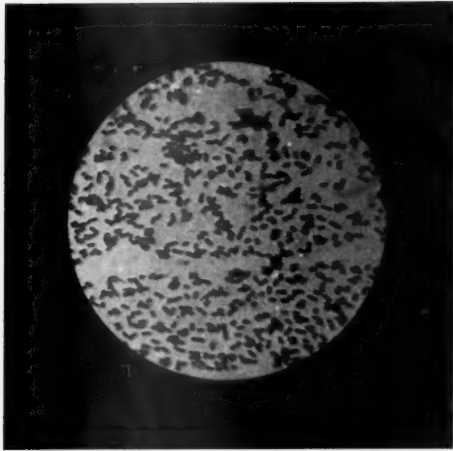
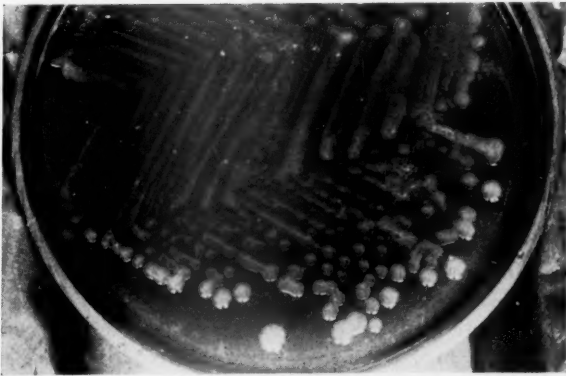Fig. 17.—*Klebsiella rhinoscleromatis.*

Fig. 18.—Bacillus of von Frisch (*K. rhinoscleromatis*) cultured in human blood agar.

DIAGNOSIS

The diagnosis of scleroma can be very easy or very difficult. It is easy for the rhinologist to diagnose rhinoscleroma in a locality where it is endemic and when the disease shows its characteristic features. It is easy to diagnose laryngoscleroma when a previous diagnosis of rhinoscleroma has been made. It is very difficult, however, even for the rhinologist, to diagnose scleroma when a similar case has not been seen before, or during the first stage of the disease, or when there is another intercurrent condition, like syphilis, leprosy or tuberculosis. Primary laryngoscleroma or tracheoscleroma are most difficult to diagnose under any circumstances. In Guatemala most cases are seen during the late stages of the disease, when the diagnosis is no problem at all. It goes without saying that no definite diagnosis of scleroma should be made, or treatment for this condition instituted, without confirmation—as far as it is possible— by the microscopic examination of a piece of biopsy tissue.

Scleroma can be mistaken for nasal diphtheria, atrophic rhinitis, syphilis, tuberculosis, carcinoma, leprosy, rhinosporidiosis, sarcoma, etc. If the possibility of scleroma is kept in mind the diagnosis should not be missed. It is not rare to see the co-existence of scleroma and another condition, like syphilis or leprosy. One of the most misleading occurrences is a positive Wassermann reaction in a case of primary laryngoscleroma.

Several complement fixation tests have been developed, but they have not proved their value in the diagnosis of scleroma.

PROGNOSIS

Scleroma has always been, and still is, considered an almost incurable condition. The treatment is intended to stop its progress, but in most cases—sometimes even after apparent cures—the disease follows its course. Radiotherapy and radium therapy have been practically the only weapons to fight scleroma with a chance of success; and even when there seems to be a cure it is, in advanced cases, at the cost of deforming scars. We have observed in two cases a spontaneous arrest of the process of scleromatous infiltration and an early change into scarred, retracted tissue, the whole phenomenon being equivalent to relative cure.

When the disease follows its regular course, the tissues may be invaded to the extent of producing horrible deformities. In the very serious cases the air passages may be obstructed in such a way, the secondary infection may be so intense, and the patient in such a state of intoxication that very easily an intercurrent disease may cause his death.

108 CASES OF SCLEROMA OBSERVED IN 1944-1948

LOCATION	M	F
Nose	33	29
Nose and upper lip	4	1
Nose, upper lip and mouth	2	1
Nose and palate	5	2
Nose and pharynx	3	2
Nose and larynx	10	4
Nose, larynx and trachea		1
Nose, palate and larynx	2	
Nose, palate and pharynx	1	3
Nose, palate, pharynx and larynx	1	
Nose, palate, pharynx, larynx and trachea	2	
Nose, mouth, palate, pharynx, larynx and trachea	1	
Nose, upper lip, mouth, cheeks, palate, pharynx, larynx, trachea and left ear	1	

Ages ranged from 8 to 68 years.

When the larynx or the trachea are invaded, scleroma frequently obstructs the airway and a tracheotomy becomes necessary. Some patients have to keep the tracheotomy tube for the rest of their lives.

Streptomycin might change the prognosis of scleroma. But it is too early to ponder its real value in the fight against the disease.

TREATMENT

It can be said that almost every chemical product and almost every surgical procedure have been tried in the treatment of scleroma. Numerous antiseptics and caustics have been applied in the hope of finding a cure for this condition; many articles have been written, praising the results of scores of different medicines, of various minor and major operations, of the use of electrocautery, diathermy, vaccines, x-rays, radium, etc. And all the efforts, all the tests, all the trials are justified because we are dealing with one of the most repulsive diseases that may attack a human being.

Some improvement, or at least some relief, can be expected from most of the measures advised by the different authors. Several of these measures act upon scleroma itself; others attack the secondary infection that is always present. Here we are going to review only those therapeutic agents which we consider might have some practical value in the fight against scleroma.

Caustics. Silver nitrate, colloidal iodine, chromic acid, trichloracetic acid and many other caustics have been used, either superficially applied or injected into the tissues. They may be of some value when associated to other more effective agents, like x-rays or radium.

Cauterization or Electrocoagulation. When there is considerable obstruction of the nose these two procedures are effective in destroying the scleromatous tissue and in opening an airway. However, if the scleromatous process is not controlled by other means the improvement is only temporary and the final result is very poor.

Mercury, Arsenic, Antimony. Some authors claim good results with intramuscular or intravenous injections of different pharmaceutical forms of these chemicals. There is so much secondary infection in a case of rhinoscleroma that it is not a surprise to observe some improvement with the use of any antiseptic. In our experience, a few days after the treatment has been discontinued the improvement disappears and the condition comes back to the starting point. However, the treatment is worth trying.

Minor Surgery. Minor surgical procedures intelligently applied can be beneficial to a patient with scleroma. Nevertheless,

the removal of exuberant tissue, the excision of nodules, the cutting of obstructing scars, etc., are advisable only when used in combination with other means like radium, x-rays or streptomycin, known to be more effective against scleroma itself. Otherwise the results are poor and the treatment does more harm than good.

Major Surgery. Serious or radical operations are, as a rule, contra-indicated during the evolution of rhinoscleroma. When, either spontaneously or as a result of the treatment, the disease has reached the stage of final fibrosis, surgical interventions may be in order. The plastic surgeon and the otorhinolaryngologist have to do whatever is possible to repair the damages caused by the disease. If streptomycin proves to be a better therapeutical agent than x-rays or radium, the field of surgery, as an after-treatment, will be very much enlarged and the cosmetic and functional results will be more satisfactory.

In laryngoscleroma and tracheoscleroma surgery has definite indications. Tracheotomy is frequently a life-saving operation; in some cases it is also performed as a preventive measure before starting intensive roentgen therapy. Laryngofissure should be cautiously resorted to; whenever possible it is best, as in the nose, not to remove the obstructing tissue until the stage of fibrosis. We have seen beautiful results after laryngofissure and removal of the cicatricial obstruction within the larynx; we have also seen a complete failure of the operation when it is performed during the first stages. Incidentally, it is good to remember that cartilage, although not invaded by scleroma, may become very soft and pliable; this is particularly true with the thyroid cartilage, a fact that should not be forgotten in order to avoid, after laryngofissure, a failure of the operation as a result of overlapping of the edges of the cartilage or the collapse of the whole structure. Laryngeal dilators should always be tried before doing any major surgery: we have seen very good results after dilatation when combined with intensive streptomycin treatment.

Obstructing tracheoscleroma may need surgical intervention, too. It should, however, be avoided whenever possible. In our experience, a good way of dealing with the soft, movable masses of tracheal scleroma is to remove them through the bronchoscope by a wide suction tube and powerful suction.

Vaccinotherapy. Autogenous vaccines have been advised in the treatment of scleroma. Whether monovalent (von Frisch's bacillus) or polyvalent, the vaccines seem to have given good results in the hands of several writers. In our experience, vaccinotherapy does

not attack scleroma itself but has some beneficial influence in the control of the local secondary infection.

Roentgen Therapy. In 1904, Ranzi reported the first two cases of rhinoscleroma treated with x-rays; the treatment had been given by Fittig. Since then x-ray therapy has been used in hundreds of cases, with comparatively satisfactory results.

In Guatemala, Wunderlich[92] was the first one to use x-rays in the treatment of scleroma and reported the first cures in 1908.

As a guide for those interested in the subject, we give here two sample techniques, described in the authors' own words.

Peter's Technique:[65]

"I use a hard radiation of a H.V.L. 1.0 mm copper, and an intensity of from 5 to 12 r/min., a field of 10/15 cm with a Holfelder cone, and give daily or every other day a dose of from 100 to 150 r measured on the patient's skin with a Mekapion up to a total dose of about 1000 r, and then after a rest period of from 6 to 10 weeks, usually 8 weeks, I repeat the series two to four times, giving, respectively, 800, 700, 600, 500 and 300 r. Oftentimes, because of the economic condition of the patient, it becomes necessary to modify the basic technic, and this is particularly true of patients who come from a long distance."

MacKee's Technique:[54]

"Only filtered radiation should be employed (3 mm Al). The dose should be within the amount required to effect an erythema—about 400 r, depending upon the age of the patient and the color of the skin. Applications are made at intervals of one month, i.e., subintensive filtered treatment. The entire face, with the exception of the nose, is shielded. An exposure is then made to each side of the nose with the target at right angles to a plane of the lateral surface of the nose. The rays are permitted to overlap at the bridge of the nose. A separate exposure may be necessary for the upper lip if this part is involved."

Radium Therapy. Kahler, in 1905, was the first to use radium in the treatment of scleroma. Since then it has been widely applied for the same purpose, all over the world. The results are similar to those obtained with x-rays. In some cases the combination of radiotherapy and radium therapy is advantageous, because they complement each other when the nature of the lesions, or their location, does not permit the correct use of either one of the two procedures. The rhinologist is of great help when radium is employed, because he can easily place the tube or needle in the right spot, and also can control the effects of the treatment. In laryngoscleroma radium gives better results than x-rays, but its correct application calls for the intervention of the laryngologist.

Peter's Technique:[65]

"Localized lesions in the nose, gums or pharynx may be taken care of by radium tubes containing from 10 to 50 milligrams. A few days after a single or fractioned dose of 80 mg-hr., filtered by 0.3 mm brass or of 250 mg-hr., filtered by 0.8 mm brass (corresponding to 300 r of hard beta or 1200 r of soft gamma

radiation), there follows definite regression of the lesion. With the higher dosage there follows a fibrinous mucositis. When this reaction has subsided, further treatment is given if it seems indicated."

MacKee's Technique:[54]

"If the nares are patulous a tubular radium applicator, suitably screened, may be placed inside the nose, first in one nostril and then in the other. The length of exposure will depend, naturally upon the amount of radium element in the tube. A 25-mg tube, screened with 1 mm brass, 0.5 mm silver and 2 or 3 mm rubber, may be left in the nose for two or three hours, in addition to the x-ray treatment applied to the external surface of the nose. If the nares are occluded the radium (tube or plaque), suitably screened, may be placed against the roof of the mouth. These cross-fire treatments, in the absence of reaction, may be administered at intervals of one month. Gamma rays or radium may be used instead of x-rays for the external irradiation. The result should be the same."

Sulfonamides. Most of the sulfa drugs have been tried in the treatment of scleroma. In our experience, they have no effect against the scleromatous process. They control, to some extent, the secondary infection while the medicine is in the blood stream; when the treatment is stopped it does not take more than three or four weeks for the condition to return to the starting point.

Penicillin. All that has been said about the sulfonamides in the previous paragraph applies to penicillin.

Streptomycin. This antibiotic seems to be a real hope in the treatment of scleroma. Streptomycin being effective against several infections caused by gram-negative bacteria, particularly those of the *Klebsiella* genus, it was logical to try it in scleroma. Blaisdell, at a meeting of the American Laryngological Association in April, 1947, reported a case of rhinoscleroma treated with streptomycin, and showed some color photographs to demonstrate that there had been definite improvement. Hara and his collaborators[41] used streptomycin in two patients in 1946 and 1947. They state that "streptomycin in adequate doses appears to be the most potent drug at our command." New et al.[62] reported a case of rhinoscleroma and laryngoscleroma treated in 1946 and "apparently cured with streptomycin."

We have used streptomycin in the treatment of scleroma since September, 1947. Twelve of the patients in our series were given the antibiotic, and in spite of the fact that the medicine has not been available in the quantities that we might have desired, we can say—as a preliminary report—that, although streptomycin is not the definite answer to the challenge of the disease, it is to date the best weapon that can be used against scleroma.

15 C. O. S.

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LX

ASYMPTOMATIC CHRONIC ENLARGEMENT OF THE PAROTID GLANDS

REVIEW AND REPORT OF A CASE

Louis Nash, M.D.

AND

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Reports and reviews of bilateral asymptomatic chronic enlargement of the parotid gland have appeared at frequent intervals in the medical literature for the past 75 years. The earlier reports are of importance primarily for their clinical descriptions and notation of incidence in relation to other well recognized diseases. More recent reports are of importance for their classifications and subdivisions of the syndrome and for a few well controlled animal experiments.

The causes of asymptomatic symmetrical chronic enlargement of the parotids may be classified as (1) inflammatory, (2) neoplastic, and (3) hypertrophy and hyperplasia. The lines of separation are not entirely distinct and sharp since many affections of the parotids have never been definitely classified as to etiology and pathology.

Inflammatory. Enlargement of the parotid may be due to a chronic, low-grade, nonsuppurative inflammation of the gland or its ducts.[5, 7, 9, 13, 15, 38, 44, 46] There are many reports of patients with persistent asymptomatically enlarged parotids who have recurring mild episodes of local inflammation, such as increased local heat and redness, mild pain, redness and swelling of Stenson's duct orifice, and a foul taste in the mouth. These attacks usually start unilaterally and then appear to spread to the opposite side. During the inflammatory exacerbations the parotid saliva contains mucus, which is normally not present and indicates a local irritation of the few mucus-secreting goblet cells in Stenson's duct. Microscopic examination of the parotid's secretion will show leucocytes and epithelial cellular debris, and cultures will most often reveal *Streptococcus viridans* in conjunction with *Staphylococcus aureus*. Sialograms show dilatation of

the smaller ductules and occasionally they will show an actual destruction of ductules resulting in a pooling of the injected opaque material similar to bronchograms in bronchiectasis. Bailey[2] reported a large series of cases of asymptomatic enlarged parotids in children whose sialograms showed this sialectasis, but in whom the saliva was clear and sterile. He believed these cases represented congenital sialectasis and he treated them with irrigations, dilatations and occasionally by parotidectomy. Payne[38] reported calcification of the dilated ductules in chronically enlarged parotids in a patient with Reynaud's syndrome and scleroderma.

Most authors believe that this type of low-grade chronic parotitis is caused primarily by a retrograde ascending infection from the oral cavity. Pyrah[40] treated such cases only by general oral hygiene and achieved good results. Other authors found their patients responded best to dilatation and irrigation of the ducts;[9, 19, 28, 34] to x-ray to the parotids;[4, 7, 13, 25] or to general antibacterial drugs such as the sulfonamides, penicillin and iodine.[1]

In many cases chronic enlargement of the parotids is definitely related to a variety of allergic phenomena.[10, 37] Usually there is some type of partial duct obstruction which aids in infecting the gland.[44] Pearson[36] reported a large series of cases in children and adolescents with an insidious onset of bilateral parotid enlargement which fluctuated only moderately in size over many years. The saliva was microscopically clear and was sterile. The sialograms showed terminal sialectasis in 50% of the cases. Three-fourths of these patients had some type of allergy such as asthma, hay fever and urticaria, and they maintained a continual blood eosinophilia. During the fluctuation in size of the gland the parotid saliva contained many plugs of inspissated mucus packed with eosinophils. Many of Pearson's patient also noted enlargement of the parotids at meal time, suggesting blockage of the duct. Frequently the fluctuation in size of the parotids would be associated with an acute upper respiratory tract infection. Pearson attributes this type of fluctuating chronic enlargement of the parotids to intermittent obstruction of the ducts due to (1) a hyperplastic overgrowth of lymphoid tissue in the duct in association with an upper respiratory tract infection, (2) an actual angioneurotic edema of the ductal mucosa, (3) muscular spasm at the oral ostium of the duct, (4) achalasia of the duct, or (5) an abnormally viscid saliva. Pearson further believes this allergic factor in chronic enlargement of the parotids to be familial since in 50% of his large series of cases there was a family history of allergy, and he also noted multiple cases of enlarged parotids in single families. He reported a case to illustrate the role of two of these

factors. This infant was observed to have rapid symmetrical enlargement of both parotids associated with intestinal colic. This could be reproduced by manually manipulating the stomach and intestines through the abdominal wall. Pearson believed this syndrome could be due to sympathetic nerve reflexes which can cause spasm of the duct ostium and also increase the viscosity of the saliva.

Partial blockage of the parotid duct by calculi has been observed in many cases of chronic enlargement of the parotids. When the blockage is complete there is a typical syndrome of sudden onset of severe pain and tenderness over the rapidly enlarging gland. When the blockage is incomplete and prolonged, the acute symptoms are absent and the gland remains enlarged. New and Harper[34] believe the acute and severe symptoms of a completely blocked off duct are due to the infection, either new or recurrent, behind the block. In most of their large series of cases of enlarged parotids they found calculi in the ducts, and they successfully treated all their patients by incising Stenson's duct ostium in the mouth (meatotomy) to establish better drainage.

Mikulicz' disease has been the subject of much confusion ever since he described the first "typical" case in 1880.[30] During the following 55 years more than 125 articles were published on this subject.[3, 4, 12, 16, 17, 22, 26, 27, 32, 42, 45] Schaffer and Jacobson[43] reorganized the entire subject in 1927, and made a distinction between true Mikulicz' disease and Mikulicz' syndrome. In a review of the older articles on Mikulicz' disease it is found that almost all of the cases reported can be classified as Mikulicz' syndrome, in which the parotid enlargement is secondary to a generalized systemic disease.

True Mikulicz' disease[21, 43] is a rare entity characterized by an asymptomatic enlargement of all, or any combination of, the salivary and lacrimal glands. The pathologic process is a characteristic replacement of the interstitial stroma by small lymphocytes. This process must be entirely localized to the salivary or lacrimal glands and have no extension to neighboring organs or tissues. The patient's general health is unaffected, although when all of the salivary glands are affected there is a dryness of the mouth (xerostomia) which results in ulceration of the oral mucous membranes and gastric disturbances.[29] When the lacrimal glands are affected there is usually dryness of the eyes with severe conjunctivitis often resulting in corneal ulcers and scars. The disease occurs with equal frequency in both sexes. The average course of the disease is 7½ years, but a case has been reported which was followed for over 15 years. There are reported cases of proved true Mikulicz' disease

limited to both parotids.[4] It has been repeatedly observed that the involved glands in Mikulicz' disease will markedly regress in size during an intercurrent severe infection or disease, but the enlargement rapidly recurs when the infection or disease has subsided. True Mikulicz' disease can be accurately diagnosed only by a biopsy of the involved gland which will show the typical histopathological picture. Only rarely does a connective tissue hyperplasia take place that results ultimately in a diffusely scarred gland.[43] The meager investigative work on the involved parotid glands reveals that the saliva is decreased in amount, is microscopically clear and contains no bacteria. Sialograms of the involved gland show a separation of normal appearing ductules from each other.[8, 24]

Mikulicz' disease is self-limiting and has no mortality. It has been treated successfully by x-ray therapy,[20, 25, 27, 33] radium,[4, 13] and penicillin.[21]

The etiology of true Mikulicz' disease evolves around the interpretation of the characteristic small lymphocyte in the gland. Is it a chronic inflammatory cell or an element of lymphoid hyperplasia? Leucutia and Price[27] believe Mikulicz' disease is a phase in aleukemic leukemia, but because of the length of follow-up on some of their reported cases it seems unlikely. They report several cases from the foreign literature in an attempt to prove the disease is an inherited lymphoid hyperplastic disease. Because of the marked difference in response to x-ray therapy of true Mikulicz' disease and other lymphoid neoplastic tissues, it is unlikely that it is a true lymphoid hyperplasia.[27] Most observers believe the disease is inflammatory in nature and caused by an unidentifiable virus. Recent animal experimentation has shown that a virus passed in utero from mother to fetus will cause a malignant epithelioma of the parotid glands of Western cotton-tail rabbits in unlimited generations. Whether Mikulicz' disease could be caused by one of these organo-specific viruses is a matter of conjecture.

The only common factor in Schaffer and Jacobsen's widely divergent group of diseases classified as Mikulicz' syndrome is the outstanding physical finding of asymptomatic chronic enlargement of the salivary or lacrimal glands or both. Each case is a relatively rare instance of the primary disease in which the predominant findings are related to the salivary or lacrimal glands. Because of the uniqueness of the syndrome it has been reported at great lengths in the medical literature.[32] Some of the diseases in which this syndrome is seen are aleukemic leukemia, leukemia, lymphomas of various types, tuberculosis, syphilis, gout, chronic uveoparotid fever, sarcoidosis and Sjögren's disease.

Chronic uveoparotid fever[5] is characterized by painless chronic enlargement of the parotids, inflammation of the uveal tract of the eye and a prolonged low-grade fever that is frequently complicated by cerebrospinal fever, polyuria, polydipsia and a generalized lymphadenopathy. Its course is from weeks to months and the mortality has been reported to be 5%.

Sjögren's disease was described in 1933 and many reports have subsequently appeared on this syndrome.[28, 49] It comprises chronically enlarged and occasionally tender parotid and lacrimal glands with reduced secretion of saliva and tears resulting in severe conjunctivitis sicca and a xerostomia. Except for the oral and eye signs the physical examination and laboratory findings are normal. Biopsies of the parotid glands reveal a picture like that seen in Mikulicz' disease. The parotid secretions show a very low amylase content. Many of the patients appear to have a disturbance of endocrine and carbohydrate metabolism.[49] The disease is stationary and chronic. Vitamin A administration has no effect on the disease, but x-ray therapy will reduce the parotid enlargement.

Zellweger[48] reported a case of sarcoidosis in a 15-year-old boy that started acutely with enlarged, poorly functioning salivary and lacrimal glands. There was an associated dysfunction of the pancreas with a low glucose tolerance curve, although the salivary amylase was high. Only much later were typical lupoid nodules of sarcoidosis found in the mucous membranes.

Chronic enlargement of the parotid glands due to selective secretion of toxic drugs has been observed for many years.[5] The intoxication is manifested by swelling of the gland, with alteration in its function and mild local discomfort. Mercury poisoning was common in earlier years because of the method of treatment then prevalent for syphilis and because of the common household use of Calomel. Lead poisoning is an infrequent disease at present because of industrial and health safety measures. The stomatitis associated with mercury and lead poisoning usually overshadows the mild parotid discomfort. Poor mouth hygiene, edema of Stenson's duct ostium, ascending infection, and allergic phenomena probably played a major part in the enlargement of the parotids. Enlarged parotids secondary to selective secretion of iodine have been recognized for many years. Recently Altemeier[1] has applied this factor successfully in the treatment of acute suppurative parotitis. Enlargement of the parotids occurring during thiocyanate treatment for hypertension is also attributed to selective secretion by the gland.[35] Parotid enlargement has been noted recently during the thiouracil treat-

ment of hyperthyroidism.[11] The cause for this response has not been determined.

Neoplasms. Rarely do true neoplasms cause a bilateral asymptomatic enlargement of the parotids. Ramage, Binnie and McCall[41] reported a case of bilateral adenolymphoma in a patient who had a palpable nodule in each gland for 15 years. They also cite a case reported by a foreign author of simultaneous bilateral mixed tumors of the parotids.

Hypertrophy and Hyperplasia. Whether there is such a disease as familial or hereditary enlargement of the parotids is an unsettled question that has appeared at intervals in the medical literature. Pearson[36] reported a study of two children in one family with bilateral chronic asymptomatic parotid enlargement. Leucutia and Price[27] report an American family with 11 persons on the paternal side with the same asymptomatic parotid enlargement. Fontoynont[14] reported a study of several hundred cases of endemic familial (hereditary) enlarged parotids from the high plateau regions of Madagascar. This disease ("Le Mangy") inflicts a great social blight on the person because of the belief among the natives as to its etiology. The disease is limited to farmers and country manual workers of the plateau region where the diet is primarily potatoes and rice. The disease affects from .5 to 2% of the population in the endemic area, but in some villages it may affect 100% of the population. The disease is primarily bilateral, the parotid gland is never inflamed or tender and the saliva is microscopically clear. The parotids are usually first enlarged during infancy, but occasionally epidemic parotitis appears to be an activating factor in the older age group and the glands maintain their large size. The sexes are equally affected and there is no evidence of any other disease. The enlargement of the parotids persists for as long as 80 years. Fontoynont presents definite familial and regional incidence of this disease but he fails to prove a definite hereditary factor. There unfortunately were no biopsies of the affected glands and no autopsy studies of affected persons. Fontoynont believes that in this region of Madagascar this disease is to the parotid gland what nodular goiter is to the thyroid.

Kenawy[23] reports a much more carefully worked up group of cases of endemic asymptomatic enlargement of the parotid glands in natives of the upper Nile Valley in Egypt. Asymptomatic enlargement of the parotids in these agricultural workers is extremely common and about 3% of all patients admitted to the hospitals in this area have these enlarged glands. Kenawy studied 100 patients that were hospitalized for various reasons and found that 10% of them had manifest or latent diabetes, 65% had pellagra and 25% had other

vitamin deficiencies and parasites. The enlargement of the gland occurs in childhood and persists for life. The disease is definitely not familial and it rarely affects women. The parotids do not fluctuate in size and are never tender. In 20% of the cases there was poor pancreatic function by Schmidt's nuclear digestion diet and one third had an abnormally high serum amylase. The parotid saliva was twice the normal amount, had a normal amylase content and was microscopically clear and free of bacteria. The saliva was completely free of thiocyanate, but this was expected in the pellagra cases. Sialograms were normal in 50% and showed moderate tortuosity and multiplicity of the ductules in 50%. Histologically the glands showed a picture of hyperplasia with the acini crowded together and an increase in the ductules, but no infiltration by lymphocytes. Autopsies on several patients showed a normal pancreas. Kenawy believed that there was probably a parotid hyperplasia to compensate for some deficiency occurring in pellagra and diabetes. He hypothesized that the absence of the normal bacteriostatic salivary thiocyanate could allow an unidentifiable virus or bacterium to cause a chronic infection in the parotid gland. He performed many experiments attempting to prove a virus agent in the disease but to no avail. During his studies, two of his patients developed acute pulmonary infections with a rapid decrease in size of the enlarged parotids. When the infection subsided, the glands rapidly grew back to their former size. He treated his patients with large doses of vitamins to no avail, but found a complete and apparently permanent regression of the large glands with x-ray therapy.

The relationship of asymptomatic enlargement of the parotids to avitaminosis has been extensively studied and reported. Fontoynont's studies[14] in Madagascar concerned laborers on a limited restricted diet. John[22] reported 9% of Polish children with malnutrition were found to have large parotids. Wenckebach,[47] in his lectures on tropical avitaminosis, noted that edema of the parotids demonstrated one of the peculiar regional affinities of the body for the anasarca fluid seen in beri-beri, and gave these people a typical broad-jawed facies. Biggam and Ghalioungui[6] reported a typical full plump facies in thousands of cases of ankylostoma anemia studied in Egypt and noted that this was definitely due to swelling of the parotids in the more severe cases. Miller[31] studied a large number of pellagra cases in Egyptian insane hospitals and found a significant percentage with enlarged parotids. He believed that the parotids hypertrophied to compensate for a pancreatic deficiency due to failure of stimulation because of the typical gastric achlorhydria.
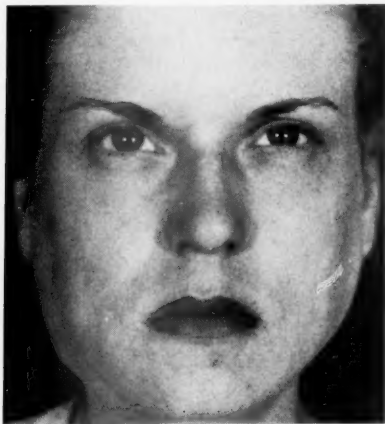
Fig. 1.—Appearance of the patient before operation, showing symmetrical enlargement of the parotid glands.

REPORT OF A CASE

This 27-year-old married white woman presented herself to the University of California Hospital Outpatient Department on September 8, 1947, with a chief complaint of difficulty in swallowing and swollen neck glands for 19 years.

Her history revealed that she had been perfectly well until the age of 8 when she simultaneously had whooping cough and mumps, with the usual swollen parotid glands. During the acute illness she began to vomit after eating and noted difficulty in swallowing solid foods. The parotid glands did not regress in size following the illness, and the enlargement persisted to the time of admission. During an episode of bronchopneumonia at the age of 22, the parotids shrank to normal size, but rapidly returned to their previous enlarged state after the acute phase of the illness was over. The parotid glands had always been soft and had never been acutely tender or inflamed. She had always secreted a copious, but no excessive amount of saliva. Occasionally she noticed gushes of fluid into her mouth from the parotid ducts. The size of the glands varied only slightly from day to day during this entire period. She believed that they were largest just before her menstrual periods, and at this time they were slightly tender and painful.

The frequent vomiting and inability to swallow solid food had also persisted. The vomiting occurred after almost every meal, and
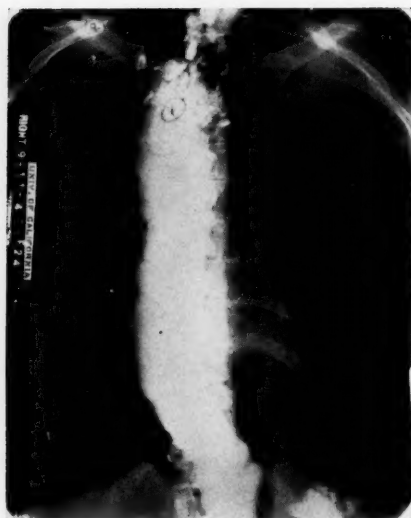
Fig. 2.—Pre-operative x-ray study shows extreme dilatation of the esophagus and narrowing of the cardia.
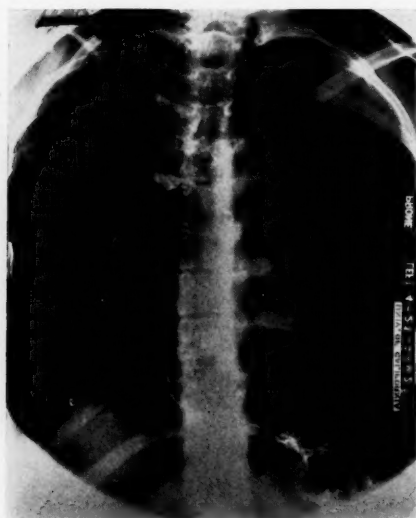

Fig. 3.—After the cardioplasty the flaccid, dilated esophagus empties rapidly.

she frequently slept half sitting up to prevent a regurgitation and welling-up of undigested food into her mouth and nose. She had a burning type of epigastric and substernal pain while eating, which was relieved by vomiting. Associated with the pain was a "gassy choking feeling" in her throat which was also relieved by vomiting.

Her condition had previously been diagnosed as cardiospasm and she had had bougie dilatations of the esophagus at the ages of 10, 13 and 16. Her swallowing difficulties and vomiting were relieved only slightly for a few weeks following each instrumentation. She had been markedly underweight since the onset of her disability.

Her past history and system review were significant primarily in a negative manner. The attack of bronchopneumonia at the age of 22 was the only severe illness she had suffered. Her use of tobacco was always very moderate. She never had any hematemesis or melena and her bowel habit was normal. The stools were always of normal color and consistency. Her menarche appeared at the age of 19 and required several "hormone" injections to make the menstrual cycle regular. She had had one pregnancy and delivered a normal child at the age of 26. A psychiatric investigation into her personality showed that she grew up in an over-solicitous home where her parents "babied and spoiled" her and tried to keep her from working. It was the opinion of the psychiatric consultant that she had a passive dependent personality that was deeply ingrained.

Her family history showed she had five siblings who were all healthy. There was no suggestive history of other parotid enlargements in her family and no evidence of functional or organic gastric diseases.

When first examined here, she was observed to be a very thin, pleasant woman whose skin showed mild acne. The parotid glands were symmetrically enlarged to about five times normal size (Fig. 1) and were homogeneously soft to palpation. No abnormalities could be palpated along Stenson's ducts, and their ostia appeared normal. Clear saliva could be seen coming from both ostia. There was no evidence of enlargement of the other salivary or lacrimal glands. The thyroid was not palpable and no other masses or nodes could be palpated in the neck. X-ray studies of the esophagus showed narrowing and distortion of the cardia and a large fusiform dilatation of the esophagus (Fig. 2). Esophagoscopy showed an absence of ulcerations and there was no evidence of tumor. A diagnosis of marked cardiospasm was made.
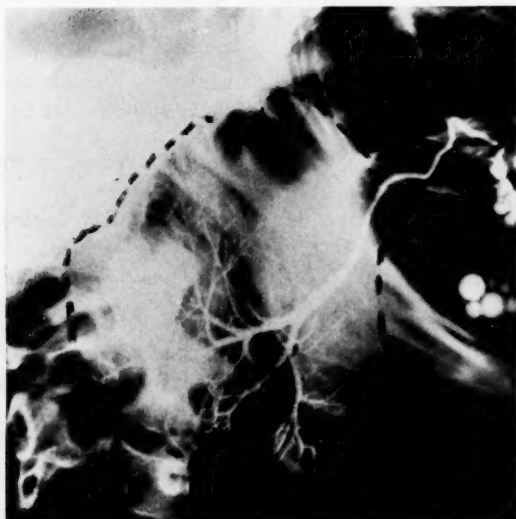
Fig. 4.—The sialogram of the affected parotid shows a great increase in size of the gland as compared to a normal sialogram (Fig. 5). Generalized dilatation and separation of the ductule system is apparent.
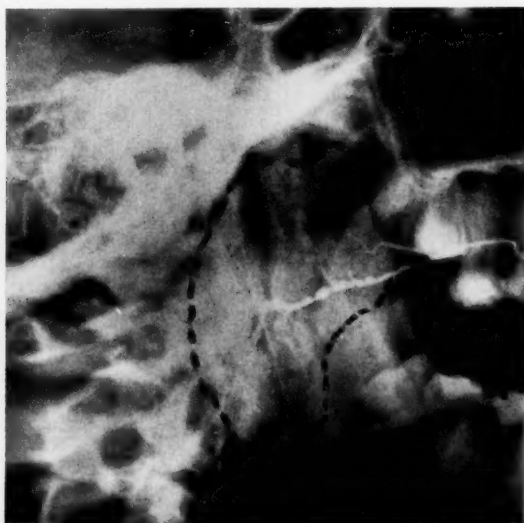

Fig. 5.—A normal sialogram obtained in the same manner as that shown in Fig. 4.

The cardia was dilated by bougies eight times in the following five months. Since dilatations resulted in only slight and transient relief of symptoms, a Finney type of esophagogastrostomy was performed. Her postoperative course was uncomplicated and she went quickly and easily through the graduated diet to a regular unrestricted diet (Fig. 3). She gained weight rapidly and had a remarkable upswing in her vitality and sense of well-being. Eighteen months after her operation she had gained 29 pounds in weight and was three months pregnant. She had had no nausea, regurgitation, vomiting or substernal or epigastric pain since her operation and found she had distress only on eating large pieces of meat or raw vegetables.

The form and function of the enlarged parotid glands were intensively investigated before and after operation and several studies of pancreatic function were performed.

Sialograms revealed a generalized uniform dilatation of the major and minor ducts with some separation of the terminal ductules from each other (compare Fig. 4 and 5). There was no evidence of ductule destruction and no evidence of duct blockage. Stenson's ducts were catheterized and the rate of secretion of the resting parotid was found to be 3.43 cc per 5 min. (normal 0.07 cc $\pm$ 2x per 5 min.). Smears of the saliva revealed an occasional desquamated epithelial cell. Cultures showed *Staphylococcus aureus* and *Streptococcus viridans*, both of which were present in swabs of her mouth. Cultures for fungus showed no growth at two months, and guinea pig inoculations were negative for tuberculosis and mycotic infection. Analysis of the saliva showed a pH of 6.0 (normal 6.0-7.9), amylase 12,568 units (normal 100-150 units), uric acid 2.8 mg% (normal 0.6-2.9). A biopsy of the parotid gland (Fig. 6) showed a very dense parenchyma with normal appearing acini and normal appearing ductules. There was a mild diffuse infiltration with lymphocytes in the periductile tissues (Fig. 7). It was believed that the proportion of acini to ductules was greater than in the normal gland.

Investigation of pancreatic function showed a blood serum amylase of 288 units (normal 80-150), blood serum lipase 0.3 units (normal 0.3-1.5), urine amylase 295 units. The intravenous glucose tolerance test showed a normal curve except for very low-fasting blood sugar levels (Chart 1). Pancreatic ferments obtained with duodenal intubation with a double lumen tube showed trypsin 27.2 units (normal 13-88), amylase 0.20 units (normal 0-0.113), lipase 0.0002 units (normal 0.002-0.0073). Chart 2 shows the
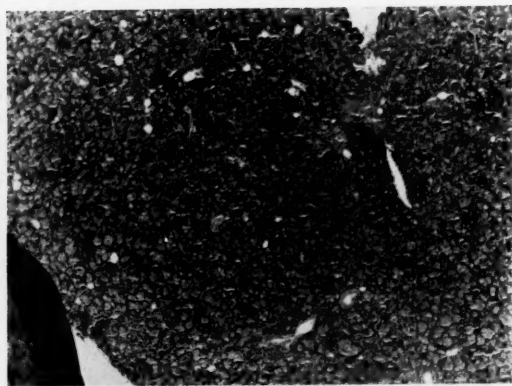
Fig. 6. —Photomicrograph of section of the parotid under low power showing very densely packed, normal appearing acini.
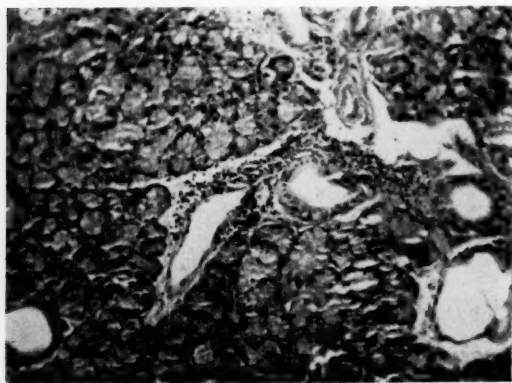

Fig. 7.—High power photomicrograph of the parotid showing mild lymphocytic infiltration of the periductile tissue.

vitamin A absorption curves using measured amounts of the vitamin before and after the administration of Pancreatin granules.

From these studies on the function of the pancreas, it was believed that there was a definite decrease in pancreatic and serum lipase with a resultant low vitamin A level in the blood which did not rise on administration of vitamin A. However, there was a significant absorption of vitamin A on the administration of Pancreatin. A biopsy of the body of the pancreas showed normal appearing acini, ducts, islands of Langerhans and stroma, without any evidence of fibrosis, inflammation, necrosis or cellular infiltration.

Incidental tests showed a normal gastric acidity and a normal gastric response to histamine. The total blood proteins were 5.79 gm% with a normal albumin-globulin ratio. Histoplasmin, tuberculin and sarcoidosis skin tests were all negative. Serum hormonal iodine was 7.3 micrograms per 100 cc (normal 4.0-8.0).

In the immediate postoperative period the patient's parotid glands regressed markedly in size and have remained at about normal size since that time (Fig. 8). Although there is still some firm, palpable parotid gland tissue, the glands are not visibly enlarged. It was impossible to catheterize Stenson's duct during the postoperative period, but the patient noted a definitely decreased amount of saliva. Ten months after surgery there was no significant change in the intravenous glucose tolerance test, the serum amylase and lipase and the salivary amylase.

DISCUSSION, SUMMARY AND CONCLUSIONS

A review of the world literature on asymptomatic enlargement of the parotid glands has been made in an attempt to gain a perspective on an entity that is common but usually not characteristic of a single disease. In view of our present knowledge of the lymphoma and leukemic neoplasms, most of the cases of Mikulicz' disease that appeared in the older literature were really only local manifestations of the generalized disease. True Mikulicz' disease is now considered a very rare disease.

Asymptomatic enlargement of the parotids in association with low-grade infection is primarily a result of the chronicity of the infection and the low virulence of the causative organism. A careful history and a few simple laboratory procedures will usually bring out the inflammatory nature of the disease. The therapy for this type of enlarged glands is usually aimed at the partial blockage of the ducts which is invariably present.
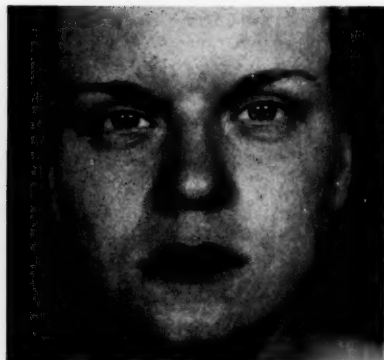
Fig. 8.—Appearance of the patient after operation. There is no appreciable enlargement of the parotid glands.

Of primary interest is the controversial problem of whether the parotid gland can increase its function by undergoing hyperplasia. This problem is apparently no nearer to being solved now than it was 25 years ago. The major evidence for this hypothesis is the well substantiated impression of numerous clinicians that there is asymptomatic enlargement of the parotids in certain phases of pancreatic disease. Definite conclusions cannot be drawn from these impressions because of the paucity of accurate laboratory and histopathological studies on the affected patients. The few recent well controlled animal experiments on the basic physiology of the parotid gland substantiate these clinical impressions by showing that there is some factor in parotid glands that affects carbohydrate metabolism. The type of factor and its method of action are unknown.

The dividing line between the chronic inflammatory enlargement of the glands and the functional hyperplastic type is not sharp and definite, and many cases seem to fall somewhere between the two classifications and demonstrate aspects of both. The reports of hereditary enlargement of the parotids and endemic enlargement of the parotids are unquestionable, but because of the locale and dietary habits of these people the probability that infective agents and marked vitamin deficiencies result in secondary functional disturbances is very strong. The action of the higher nerve centers on parotid function has been set down as a dictum from the basic studies on reflex arcs and observance of the gland function in infancy, old age
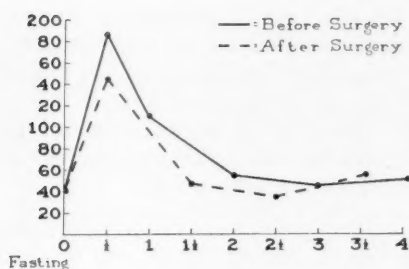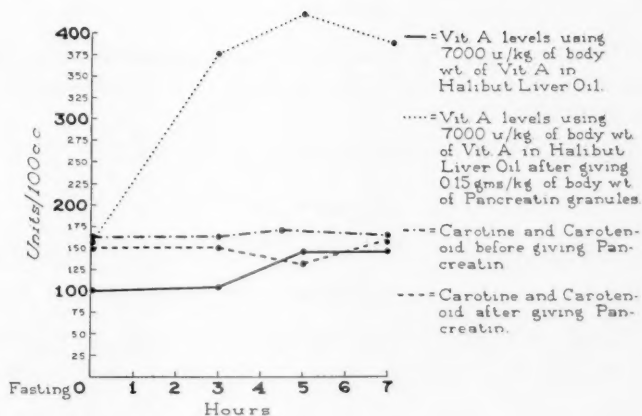
CHART 1

CHART 2.

and in degenerative diseases. The concepts of the psychosoma are interesting but difficult to prove.

A case is presented of a young woman who had asymptomatic bilateral enlargement of her parotid glands and a severe cardiospasm for 19 years. Investigation of the parotids showed a markedly increased function without evidence of inflammation. Parotid biopsy showed hyperplastic glandular tissue with no evidence of neoplasm. Investigation of the pancreas revealed a normal glucose tolerance curve with low fasting blood sugar levels and practically absent lipase ferment. The parotid glands returned to normal size after surgical excision of the stenosed cardia.

Could there be a single causative factor for disturbances of her parotids, cardia and pancreas? The perusal of the literature shows this association is not uncommon. Possibly a general vagotonia had persisted since childhood. Perhaps the syndrome was a secondary manifestation of cerebral damage by the mumps virus. The regression in size of the parotids following the opening of the cardia suggests that the parotid hypertrophy and hyperplasia were secondary to the pooling of decomposing food in the obstructed esophagus. In other words, it was a normal response of the body to wash the noxious material from the esophagus.

The nature of this syndrome remains undetermined and it brings again to the forefront the need for further investigation of basic physiology.

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THE FACTOR OF HEREDITY IN LABYRINTHINE DEAFNESS AND PAROXYSMAL VERTIGO (MÉNIÈRE'S SYNDROME)

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As antibiotic therapy marches on, hereditary disease forms a larger and larger proportion of medical practice. There have been three chief methods of determining whether a disease belongs in this category: (1) More than one instance of an abnormality in the same or different generations of a family; (2) in several offspring of a consanguineous marriage; (3) in both identical twins.

My suspicion that labyrinthine deafness and paroxysmal vertigo were inherited was first aroused when two brothers were seen in the Out Patient Department of the Massachusetts General Hospital. Soon afterward a father and son, a father and daughter, a brother and sister and two sisters were encountered either in hospital or office practice. This suspicion was reported in 1941[1] and was strengthened in 1943 by finding three affected siblings resulting from a consanguineous marriage. Nineteen forty-five brought identical twins with labyrinthine deafness and, to date, one with paroxysmal vertigo and vomiting.

TWO FAMILY CASE HISTORIES

CASE 1.—Two sisters and a brother in one French Canadian family had suffered from deafness and paroxysmal vertigo. The parents were first cousins, and two maternal uncles had one son each with this disorder (Fig. 1). There was no history of deafness in the parents or their siblings. The two sisters were aged 46 and 32 respectively at the onset of their vertigo. The first sister had been admitted in February, 1941, to the Massachusetts General Hospital following an attack of vertigo, vomiting and diarrhea on awaking in the morning. She had noticed tinnitus in the right ear for two years. An internist, neurologist and otologist agreed on the diagnosis

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First cousins

● affected
○ unaffected
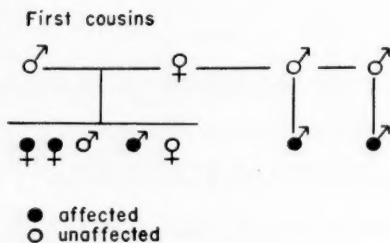
Fig. 1.—Family tree of a consanguineous marriage.

of Ménière's syndrome, and an audiogram was ordered (Fig. 2). She was put on potassium chloride 25%, two teaspoons three times a day, and discharged. She discontinued this in four months' time and was well until September, 1943, when she had another attack of vertigo and vomiting. There were two severe attacks in November accompanied by great roaring in the right ear in spite of her return to the 6 gm potassium chloride per day. In the last attack she had fallen to the aisle floor of a crowded commuters' train and had vomited. She was sent to me in December, 1943, and was placed on 2 gm 25% potassium chloride six times a day and asked to limit salt intake. She was well until November, 1945, when she had several severe attacks. She again returned to the potassium chloride and had improved when last seen in December of 1945. Her sister who had complained of the same symptoms in the past accompanied her to the office. On examination both sisters complained of tinnitus and had bilateral perception deafness, more marked on one side than the other. The second sister had been treated with a low salt regime and 6 gm potassium chloride per day with relief of the acute attacks of vertigo and improvement of both tinnitus and hearing. The brother, who is a missionary, wrote from northern Rhodesia that when he was 35 years of age he heard a small buzz in his right ear. One day while walking, the sidewalk "came up" to meet him. This was followed a few days later by emesis and vertigo. There was no pain in that ear but some tinnitus. Section of the eighth nerve relieved the condition. The sister stated that two of her first cousins had had symptoms similar to her own. One of them, a druggist, had followed the treatment outlined for the patient and had secured relief from his paroxysmal vertigo.

CASE 2.—In July, 1945, identical male twins, aged 47, were seen in the Neurological Out Patient Department of the Massa-
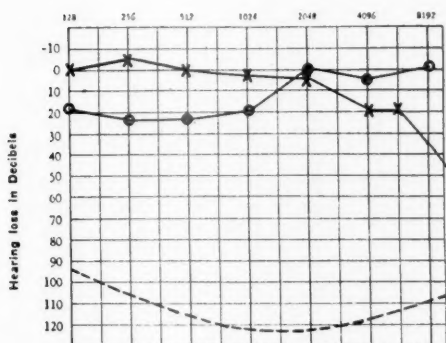
Fig. 2.—Audiogram of one of three siblings of a consanguineous marriage. Right: O; left: X. Roaring in right ear. + Rinné in right ear. Weber lateralized to left.

chusetts General Hospital. The first complained of sudden increase of deafness on the right, tinnitus and paroxysmal attacks of vertigo and vomiting. Up to the present time the second has had non-progressive deafness unaccompanied by vertigo. The first twin had noticed deafness and tinnitus at the age of 31, the other at the age of 35. Neither had had otitis media in childhood, and the family history revealed that one aunt had been deaf. Both had marked perception deafness on the right and some on the left (Fig. 3). The attacks of vertigo and vomiting in the first twin were controlled with some difficulty. On one occasion, while walking on the street, he fell to the sidewalk and vomited. Only after admission to the hospital in March, 1946, and after the dose of potassium chloride had been temporarily increased to 12 gm per day were his attacks controlled. When last seen in October, 1948, he was wearing an air receiver hearing aid, still complained of tinnitus but had had no attacks since March, 1946.

Pathology. In 1908 and in 1913 "ectasia of the ductus cochlearis, saccularis et utricularis" or hydrops of these portions of the membranous labyrinth was related to a single attack of deafness, tinnitus and vertigo[2, 3] (serous labyrinthitis), but only in 1941 was this defect given a causal relationship to long-continued labyrinthine deafness and paroxysmal vertigo (Ménière's syndrome).[4] Lindsay, in 1944, published similar plates of the dilated membranous labyrinth and made the very important point that vertigo is not always, and probably much more often not, a symptom due to this pathology.[5]
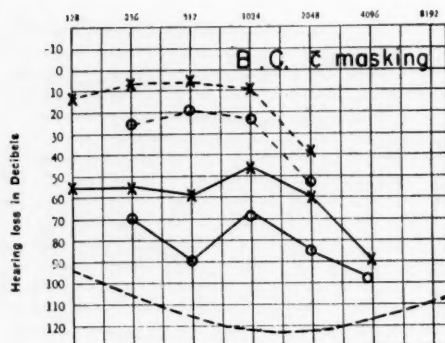
Fig. 3.—Audiogram of one of identical twins. Right: O; left: X. Weber not lateralized with 128 fork. Rinné negative. Weber lateralized to left with 512 fork. Bilateral tinnitus, more on left.

Labyrinthine deafness and tinnitus only may accompany the changes in endolymphatic pressure. In fact, ectasia of the cochlear duct might be found at autopsy just as is otosclerosis in patients who have not complained of deafness in life. Why is some loss of hearing and mild vertigo permanent? Is there damage to the organ of Corti and the maculae of the saccule and utricle?

Familial Deafness. Of 16 patients with deafness and paroxysmal vertigo questioned 7 had one parent with early and severe deafness.[1]

Familial Sick Headaches. It was apparent that migraine headaches appeared more frequently than is usual, in the past histories of patients with Ménière's syndrome. This fact led to questioning concerning the family history of paroxysmal headaches and vomiting.[1] Of 16 patients questioned 9 said one parent had suffered from migraine headaches and 3 had had sick headaches themselves. Paroxysmal sick headaches are hereditary in origin, occur in about 5% of the population and usually commence in childhood.

Discussion of Hereditary Deafness. Otosclerosis to date has held the center of the stage as a type of hereditary deafness, and it is not surprising that medical literature contains many instances of otosclerosis in identical twins.[7] The older textbooks mentioned vertigo as an unusual symptom of otosclerosis, but did not mention paroxysmal vertigo and vomiting.[8] Shambaugh in 1933 described male and female identical twins with perception deafness.[9] He men-

tioned the possibility of otosclerosis with nonfixation of the stapes, but from recent studies of temporal bones there is no reason to believe that otosclerosis plays a part in labyrinthine deafness.

Discussion of Labyrinthine Deafness Alone and Labyrinthine Deafness with Vertigo. Apparently Reissner's membrane of the cochlea becomes stretched under pressure from paroxysmal excess endolymph formation, and deafness and tinnitus result. If the membranes of the saccule and utricle become severely affected, the patient complains of vertigo. The defect is often bilateral and more marked on one side than on the other, but the membranous labyrinth of the semicircular canals is not affected,[4] perhaps because it is a heavier and better supported membrane than that in the cochlea, saccule and utricle. The excessive amount of endolymph is thought to be vascular in origin. One bit of evidence that patients with Ménière's syndrome have poorly regulated blood vessels is the large percentage who have had migraine themselves or who give a family history of migraine. Ménière's syndrome is most commonly a disease of early middle age, but in two patients not included in this study the symptoms commenced at 17 years. The younger the person afflicted with labyrinthine deafness and tinnitus, the more likely is he to suffer from paroxysmal vertigo.[6]

SUMMARY

During the past eight years postmortem material has revealed that in Ménière's syndrome the membranous labyrinth of the cochlea, saccule and utricle is dilated. This is true whether labyrinthine deafness and tinnitus only are present, or both deafness and paroxysmal vertigo.

Five families in which two members suffered from this syndrome have been reported. In addition, affected members of two more families are herein described: three siblings, children of first cousins, and identical twins.

The frequent family history of deafness and paroxysmal sick headache and their possible relationship are discussed.

In conclusion, labyrinthine deafness and paroxysmal vertigo appear to be inherited disorders.

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LXII

PERENNIAL NASAL ALLERGY

A REVIEW OF EIGHTY CASES

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In the management of the diseases of the nose and paranasal sinuses, it is generally conceded today that allergy is the most important etiologic factor. The following report is presented with the idea of emphasizing some pertinent points in diagnosis as well as in otolaryngologic and allergic management.

For this study 150 case records were selected from our files. The patients were requested to report for examination, or a detailed questionnaire was sent to their last known address. A total of 84 were contacted, and of these the first 80 will constitute the material for this report.

In this series of patients there were 7 (8.75%) who had symptoms of 20-30 years' duration, 8 (10%) of 15-20 years, 11 (13.75%) of 10-15 years, 24 (30%) of 5-10 years, 20 (25%) of 2-5 years, and 10 (12.5%) of 1 year's duration. Of the 80 patients, 16 (20%) had been under allergic management for 1 year, 28 (35%) for 2-5 years, 24 (30%) for 5-10 years and 12 (15%) for 10-15 years. Of these patients, none had been under allergic management longer than 15 years.

A careful and detailed history is essential in the study of an allergic individual. It is my opinion that a questionnaire given to the patient to be filled out at home, prior to taking the detailed history, is a valuable aid as it assists in making the patient "allergy conscious," and, consequently, a more "pointed" history can be obtained in much less time. We concur with the opinions of King,[1] Rawlins,[2] Thacker[3] and others that a careful, detailed history is extremely important, as or more so than skin tests.

The symptoms of patients in this series and their order of frequency is shown in Table 1. Nasal obstruction (60%) is bilateral, constant, or may alternate from side to side. Obstruction

TABLE 1
SYMPTOMS IN ORDER OF FREQUENCY

	No.	%		No.	%
Nasal obstruction	48	60	Itching eyes	12	15
Symptoms increased at night	33	41.25	Joint pain	11	13.75
Sneezing	32	40	Sinusitis (Self-diagnosis)	10	12.5
Postnasal drip	32	40	Indigestion	6	7.5
Fatigue	26	32.5	Skin manifestation	6	7.5
Cough	24	30	Anosmia	5	6.25
Headache Around eyes 11 Generalized 6	23	28.75	Ear	5	6.25
Asthma	16	20	Smoke	5	6.25
Constant or recurring colds	12	15			

confined to one side is usually not of an allergic nature. Headache occurred in 23 (28.75%) patients; in 11 it was located superficially about the eyes and in 6 it was generalized deep in the head, the latter probably being a physical or intrinsic allergy as described by Williams.[4, 5] Ear symptoms were noted in 5 (6.25%) patients, 4 of whom showed a slight conductive deafness (one patient had more than fifty tubal inflations). One patient with Ménière's syndrome was relieved by nicotinic acid. Intolerance to tobacco smoke was reported by 5 (6.25%) of these patients. It is of interest to note that Coca[6] reported instances of nonreagenic sensitivity to smoke characterized by dizziness, headache and constipation, which disappeared when the individual discontinued smoking. In this series one patient with a history of constipation was relieved of this symptom. Skin tests for tobacco smoke were not performed.

Among the patients listed in Table 2 the nose appeared normal in 31 (38.75%). The appearance of the mucous membrane and turbinates, however, is often diagnostic. The presence of an excess of mucus, the pale or purplish boggy membrane with beginning polypoid degeneration of the turbinates, polypi, singly or in combination are important. Nasal smears must be taken and stained (Hansel's quick polychrome stain) for eosinophils. Using the cri-

TABLE 2
NASAL EXAMINATION

	No.	%		No.	%
Essentially normal	31	38.75	Polypi	8	10
Excess mucus	29	36.25	Polypoid degeneration of middle turbinate	5	6.25
Hypertrophy	11	13.75	Eosins in smear + or better	53	66.25
Boggy membrane	9	11.25	Diagnostic antral punctures with eosins	8	10
Pale membrane	22	27.5	Purple membrane	5	6.25

teria of 1+ or more eosinophils (Hansel[7]) for a positive diagnosis, 53 (66.25%) positive diagnoses were made. We feel certain this number would be greater if additional smears had been examined, especially among those with superimposed infection. In those instances in which the x-ray films show the antra to be 4+ clouded, we believe that a diagnostic antral lavage should be done in the region of the natural ostia and the secretion obtained stained for eosinophils. If the patient is allergic the smear usually shows a marked predominance of eosinophils (3 and 4+). This proved to be true in 8 (10%) of the cases which were studied.

X-ray films of the paranasal sinuses were normal in 16 (20%) patients; clouding occurred in 28 (antra, 24; ethmoid, 14; frontal, 4) or 35%. In our experience the x-rays have not been diagnostic in the differentiation of allergic and infectious sinusitis. It is reasonable to assume that if the mucous membrane of the nose is edematous the membrane of the sinuses is also edematous and can thus cause the increased density found on x-ray examination. It is not infrequently noted that normal appearing sinuses may be observed by reray 48 hours after the first x-ray film has been taken.

The total number of surgical procedures in this group was 18. Of these, 11 surgical procedures were carried out before allergic management was instituted and following allergic investigation. In one patient who had had an intranasal ethmoidectomy and submucous resection a polypectomy was performed after allergic management was instituted. The most radical surgery was instituted during the era when all hyperplastic sinusitis was considered primarily the result of infection. In those instances in which radical surgical procedures had been instituted previously, with resultant

TABLE 3
ANALYSIS OF SURGERY PERFORMED AFTER
ALLERGIC MANAGEMENT INSTITUTED

	SURGERY	%
Foods	Antra	100
(Proven by trial)	Septa	100
Inhalants		
Foods and Inhalants	Intranasal	
without	Antra	75
Antihistaminics	Ethmoid	
Foods plus		
Antihistaminics	Polypectomy	100
Inhalants plus		
Antihistaminics	Septum	75
Foods and Inhalants	Antra, Ethmoid, Septum	0
plus Antihistaminics	Antra, Septum, Polypi	75

%=Total relief of allergy by allergic management plus surgery.

structural damage, a satisfactory result was difficult to obtain in spite of adequate allergic management. In this type of patient, with patent sinuses and recesses in the nose, severe exacerbations occur following acute infections. This necessitates a great deal of local therapy and the use of antibiotics. In this group three patients had had external ethmoidectomies, radical antra and submucous resection. Two of them had had turbinectomies; two had radical antra, intranasal ethmoidectomies, submucous resection and turbinectomies. In contrast, there were seven procedures (Table 3) performed after allergic management. There were no turbinates sacrificed, nor were there any external ethmoidectomies. The rather large number subjected to surgical intervention can be explained by the fact that this vicinity (Ohio River Valley) is known as a "sinus belt" with a high incidence of acute upper respiratory disease and infectous sinusitis. Sinus surgery is to be avoided, if possible, and, as Rackemann[8] so ably stated, "Sinus surgery still remains as a difficult and very troublesome problem."

Eighteen (22.5%) patients were found to be sensitive to food alone (Table 4), as proven by trial. It is advisable in food-sensitive patients to use an elimination diet only, if the allergen is one or more of the common foods, as elicited by skin tests, even though the pa-

TABLE 4
RELIEF OBTAINED BY ALLERGIC MANAGEMENT

	NUMBER OF PATIENTS	100%	75%	50%	25%	0
Foods (Proven by trial)	18	10	5	3		
Inhalants	12	8	4			
Foods and Inhalants without Antihistaminics	12	4	4	3		1
Food plus Antihistaminics	8	4	3	1		
Inhalants plus Antihistaminics	16	4	10	1		1
Foods and Inhalants plus Antihistaminics	14	6	4	2		2
TOTALS	80	36	30	10		4
PER CENT		45%	37.5%	12.5%		5%

tient reacts to inhalants. We believe this explains the high percentage of pure food sensitive patients in this group.

Although the percentage of pure inhalant sensitivity is rather low in this series of perennial nasal allergy cases, the results have been gratifying. Removal of the patient from his surroundings (cattle, dogs, etc.) often has effected a cure. Furthermore, treatment with house dust and molds has been most gratifying. The low dosage "optimum dosage" was used in the majority of cases. (Gilford and Frank[9] found that clinical results did not depend on high blocking antibody titer.) In short, the more severe the clinical symptoms the smaller should be the initial dose, and it should not be increased when relief of symptoms has been obtained. When this occurs the treatment-interval should be increased. Most of these patients were treated with house dust containing 10% alternaria, which is very prevalent in this vicinity, even though the skin test was negative. In some cases it was necessary to increase the dose to the more concentrated solutions (Sprague and Aaron[10]).

Antihistaminic preparations are appearing on the market at an alarming rate. We have been informed that at present there are 17

products in use and that by January 1, 1949, 25 preparations can be expected to be available. Numerous papers[11-17] have appeared in the literature in the past two years, pointing out their efficacy in allergic disease and also acute upper respiratory infections (Brewster[18]). Dosages and side reactions have been well worked out. However, due to wide publicity, people are using them indiscriminately and prescribing for one another. One patient had taken five Benadryl capsules a day for three years for nasal obstruction, with resulting hypertension. Antihistaminics are a valuable adjunct to therapy but definitely are not curative and should be used during the severe, acute exacerbations only, not daily nor while testing (Harley[19]).

Patients were asked in our questionnaire whether humidity increased or improved their symptoms. Only 19 answered the question, but of these, 18 (22.5%) stated that humidity increased their symptoms while 1 (1.25%) reported improvement. Similarly, air-conditioning caused an increase of symptoms in 15 (18.75%) patients; in 6 (7.5%) it resulted in improvement. Twenty-three (28.75%) patients stated they were free of symptoms in the southwestern states, 9 (11.25%) were improved and 9 (11.25%) were not affected by the change of climate (Fabricant[20]). Symptoms of 29 (36.25%) of the patients were increased by emotional stress, which is a combination of physical and antigenic allergy.

Vitamin therapy, especially treatment with large doses of ascorbic acid, has had its popularity. Newbold[21] noted the plasma levels of ascorbic acid at the time of skin testing. Then he gave large doses of ascorbic acid but found that they had no effect on the skin reaction produced by the intracutaneous injection of ragweed extract. Ruskin[22] states that experimental evidence shows that vitamin C has a synergetic action on adrenalin, causing about twice the bronchodilating effect as adrenalin alone and a much more rapid histaminic autogenesis. He found it of definite value in the treatment of hay fever and asthma. In the series presented here, 10 (12.5%) patients felt that vitamin therapy was of value.

Vaccine therapy was instituted in a series of 8 (10%) patients, two of whom stated they were benefited. The question of allergic sensitivity to invading bacteria is a moot question. Scherago[23] stated many allergists are unwilling to accept hypersensitiveness to infecting bacteria to explain such conditions as asthma and hay fever, because of the delayed and inflammatory skin reactions which are obtained by bacterial products, instead of immediate urticarial wheal, as obtained with simple protein extracts. Also, he states,

they indicate that general reactions with urticaria and coryza rarely are produced with bacterial vaccines. Other allergists believe that when a patient has one or two attacks of asthma during the year, with no change in environment, and when there are acute respiratory infections definitely associated with the attacks it is justifiable to diagnose the condition as "bacterial asthma" and consider the infection responsible for the bronchial spasm. We believe the latter to be true, probably because there has not been too much systematic work done on the subject. Shambaugh[24] states there are two kinds of chronic sinusitis; first, pure infection due to anaerobic type of streptococcus; and second, allergic with various types of secondary invaders. Stoesser[25] studied 214 children with allergic rhinitis, bronchial asthma, or both, with definite histories of infection. Fifty-nine did not respond to allergic management and surgery. These were skin tested with bacterial protein but tests were not consistent. These patients were treated with autogenous and stock vaccines. This therapy was found to be of little value except in those who were treated with undenatured bacterial antigen. In the series presented herein two patients with asthma were improved after autogenous vaccine therapy.

Penicillin therapy was used in a total of five patients in this group. Four stated there was no improvement, and in one patient, an asthmatic, the improvement was reported as 75%. Schenck[26] found that secondary infections of allergic tissue are less amenable to antibiotic therapy than infection in normal tissue. On the other hand, Schonwald and Deppe[27] found penicillin of definite value in patients who did not respond to antiallergic therapy. Nine (11.2%) patients were found to be drug-sensitive, the most common offender being aspirin. To the question as to whether their symptoms were affected by their nutritional state, 29 (36.2%) patients answered in the affirmative. If they gained weight and were feeling well their allergic symptoms were absent or diminished. If, on the other hand, they lost weight, fatigue increased and their symptoms increased in severity. We agree with Rawlins[2] that a skilled nutritionist should be consulted to correct dietary deficiencies and add essential minerals, vitamins, etc., if the best results are to be obtained from dietary regime.

SUMMARY

1. An analysis of 80 cases of perennial allergic rhinitis has been presented.

2. On the basis of the clinical history, the rhinoscopic examination, the study of the nasal secretions and the x-ray examination, the otolaryngologic diagnosis may be readily established.

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3. In most instances, it is advisable to defer all surgical procedures until allergic studies and management have been instituted.

4. A detailed clinical history of the patient from the allergic standpoint is most important.

5. Skin tests must be properly performed and evaluated.

6. Etiologic factors should be eliminated or avoided when possible.

7. Drugs should be used only as absolutely needed.

8. Food factors must be evaluated.

9. Immunization therapy for inhalants and pollens has proved to be most satisfactory.

10. The general management of the patient from other standpoints, paying particular attention to the nutritional status, is most important.

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LXIII

PRACTICAL ASPECTS OF A SUCCESSFUL SCHOOL HEARING CONSERVATION PROGRAM

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When this program was started in 1935, there was very little precendent to follow. A few American cities had been doing some phonograph screening tests in their schools. In my city, we had had screening for about seven years but the efforts stopped there. The results left much to be desired. During these past 14 years, it was a question of trial and error. Many more methods were tried and forgotten than there are steps in our present program. Although we think our present system is quite good, we are constantly trying out new points here and there in order that we might improve the system as presently in use.

I shall describe the program that is in use today. The practical points that have made it work will be stressed. The nature of this hearing conservation program is such that the essential points could be adopted in communities smaller or larger than Cleveland which has a population of about 1.5 million. Finally, I shall show you our results of which we are quite proud.

In America, we feel that such programs work more efficiently if they are controlled at the local community level. This local control is in the hands of the same people who have charge of all the other school problems. It is completely divorced from all other local government activities. The financial problem involved is of little importance, particularly, if the most important person, the otologist, is willing to take a goodly part of his pay in prestige and experience.

A good hearing conservation program must have the following points:

I. Screening tests.

    A. Group testing with phonograph audiometer.

        1. Fading numbers record.

        2. Similar words record.

B. Sweep check testing with pure tone audiometer. (For the past five years, we have used this method.)

II. Complete individual otological examination in all suspected cases.

III. Classification and disposition of cases.

IV. Adequate follow-up methods for all recommendations.

The selection of cases for screening and the preliminary arrangements are the same no matter which method is used. Principals, assistant principals, teachers, and nurses must be fully aware of the importance of such a screening program, and sufficient advance notice must be given these people. At least three weeks before a school is screened, the principal receives a form notice from the audiometer technician refreshing her mind as to which cases are to be screened and the day of the screening. This notice asks the principal to advise the technician if, for any reason, this particular day is not satisfactory.

The following children are screened:

1. All children at the eight-year age level.
2. All children at the twelve-year age level.
3. Any child that is known to have, or thought to have had, ear disease.
4. Any child that the principal or teacher thinks may be a hearing problem.

When the audiometer technician arrives at a school, a conference is held with the principal and the nurse, if she happens to be in the building on that day. It is very helpful if the schedule can be arranged in order that the nurse is there on that day. A quiet inside room on the top floor of the building is selected. This room must have adequate ventilation and be equipped with an electrical outlet for the audiometer. The school nurse or another selected individual can act as monitor.

The children are lined up outside this room and given a brief résumé as to how this test is carried on. Each child carries his own health card. There are two children in the room with the technician at all times. One child observes while the other is being tested. The right ear is usually tested first and the audiometer set at 15-db loss. The frequency dial of the audiometer is changed rather quickly from 256 to 4096 inclusive. No signal or push button switch is used. The child's face is constantly observed and he is asked to nod when

he hears a new tone. The child then places the ear phone on the opposite ear and the frequencies are changed from 4096 back down to 256.

If the technician is satisfied that this child has heard all these frequencies, the health card is stamped showing the date and the fact that a pure tone audiometer test was given and the child's hearing found to be within normal limits. The child that was observing sits down, and a new observer is admitted.

If the child does not seem to hear some frequencies and it requires increased intensity up to 25-db loss or more at two succeeding frequencies, the child's name is written on a piece of paper and his health card is not stamped.

After the first screening in that particular school has been completed, those children who were not stamped as normal are retested in the same manner. If this child is still found to show a possible hearing loss his health card is stamped indicating that the child has been referred to the otological clinic. At the end of each day's screening, the audiometer technician makes out a list in triplicate of these children suspected of having a hearing loss. The first list is forwarded to the nurse in charge of the otologic examinations. In Cleveland, this is the nurse at our center of testing, Alexander Graham Bell School. The second list is given to the school nurse at that school and the third list is given to the principal of that school. The school nurse has these children checked by the school physician for the presence of an appreciable amount of cerumen in the ear canals. One of the child's parents is then called to that school and the situation is explained to the parent. With the parent present, the otologic examining center is called and an appointment is made that is satisfactory to all concerned. At that time it is impressed upon the parents that they must keep this appointment with the child. If the school physician has found cerumen in one or both of the ear canals, the parent is advised that this must be removed before the otologic examination can be done. The school nurse then completes an Otologic Consultation Blank and forwards it to Alexander Graham Bell School. This blank contains such pertinent facts as to how this case was selected for further examining, the level of school work the child is doing, results of any psychological testing, and other serious ailments. The system has been developed to such a point that less than 2% of these children fail to be examined at the Otological Clinic.

The essential points in a complete otological examination are:

I. History.

    A. Dates, character and length of ear discharge.

    B. Frequency of earaches.

    C. Types and effectiveness of therapy.

    D. Patient's, teacher's and parents' opinion of hearing.

II. Nose, ear and throat examination.

    A. Ear drums and nasopharynx minutely.

    B. Nose and throat casually.

III. Audiometer tests.

    A. Complete pure tone air conduction.

    B. Pure tone bone conduction if indicated.

    C. Speech tests.

Early in the development of this program, one of the big stumbling blocks was the inability to convince the parents that we were dealing with a serious situation. This was doubly difficult if the parent took the child to a physician who, after a wholly inadequate test, told the parents that the hearing was normal. This has been completely eliminated by having a parent present at the examination. By allowing the parent to hear one or two frequencies which the child very positively says that he can not hear, the selling job is completed. The most positive person in the world can not unsell this idea and, furthermore, the parents will then be willing to do something about the condition.

Now that we have diagnosed the hearing situation of this particular child, the next step is to classify the case and advise about the disposition of the child. Because the writer has been opposed to the use of the terms deaf and hard-of-hearing, the use of groups has proved to be more effective. These groupings are as follows:

Group No. 1. Cases with no hearing and cases with hearing of no more than 35% in the better ear. These children are permanently transferred to a special school (Alexander Graham Bell).

Group No. 2. Cases with a probably permanent impairment and with hearing no better than 55% in the better ear. These children are transferred to one of the three Hearing Conservation Centers. Except for athletics and other group work they are under a speech reading teacher and use a group hearing aid all day.

Group No. 3. Cases with a potentially remediable impairment and hearing no better than 70% in the better ear. These children are transferred for from one to three years to a Hearing Conservation Center and are under a speech reading teacher about one and one-half hours per day. The rest of their time they are in regular classes.

Group No. 4. Cases with repeated temporary impairments and cases with hearing no better than 85% in the better ear. These children receive about one and one-half hours of auricular training each week.

Group No. 5. Cases with an occasional temporary impairment. Nothing special is done in these cases.

After the case has been diagnosed and classified into its proper group, the findings and recommendations are recorded on the consultation blank that had been forwarded to Alexander Graham Bell School. This blank is then returned to the school from which the child was referred. It is then permanently attached to the child's health card. If the case has been classified into group No. 1, 2, or 3, the parent and child are given an immediate interview by the principal of Alexander Graham Bell School. If the recommended transfer is agreed to, the arrangements are made at this time. If the case is classified into group No. 4, the parent is given an instruction slip advising him or her as to how they may contact The Cleveland Hearing and Speech Center. At the same time, the director of this center is advised about this case. When the child is enrolled in one of their classes this notification slip is destroyed. Until it is destroyed, the parents are contacted again and again until the child becomes enrolled. In these milder cases, one or two home calls by the social worker are sometimes necessary.

There are several types of cases that are put on what we refer to as our "call-back cards." These cases are as follows: Active otitis media in one or both ears, potentially improvable hearing impairments for which medical treatment has been recommended, children that have been classified in either group No. 1, 2, or 3, but whose parents have not consented to the transfer at that time. We have a "call-back" card for every month in the school year. We can arrange to call the children back in three, six, or twelve months, depending upon the circumstances.

In the table one can see the comparison figures during the past 14 years. In the school year of 1935-1936, the number of cases screened was over 21 thousand and the number of suspected cases was 6.7% of the total. During the past school year, the number of

suspected cases was only 307 or 2.1% of the cases screened. Over the 14-year period there was 4.3% of all children tested that had a suspected hearing impairment. In the first year of this program,

COMPARISON OF FIGURES FOR 14 YEARS—1935-1949

	1935-1936	1948-1949	TOTAL
New cases screened	21,815	14,291	207,082
Suspected cases	1,465	307	8,824
Percentage	6.7	2.1	4.3
Complete ear exams	92	442	3,653
Percent of suspects	6.3	143.9	
Known running ears	622	37	

(This represents a reduction by 94%.)

Average school population: 165,000

there were only 92 complete ear examinations done while last year there were 442. The fact that during last year, something over 140% of the suspected cases received complete examinations is explained on the basis that as the system has progressed many other agencies now refer cases in addition to our own audiometer screening technicians. The figures concerning active otitis media prove conclusively that such a program is worth while. The first year of this program, we discovered 622 children with active otitis media. Last school year, we know that there were only 37 such cases in this school area. This represents a reduction of 94% which is an accomplishment that truly evaluates such a program. Statistics on the reduction in the number of pupil failures with consequent saving in educational costs are not as accurate as those on otitis media. However they do show that one-half of these hearing problems failed at least one year. Now, the percentage of failures among these hearing-problem children is less than the over-all percentage.

10515 CARNEGIE AVENUE.

LXIV

THE PRESENT POSITION OF FACIAL NERVE SURGERY

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Sir Charles Ballance and Dr. Arthur Duel,[1] friends and colleagues for 30 years, dissatisfied with the results of anastomosis operations for the relief of facial paralysis, began in 1930 in this country a series of animal experiments. These researches, conducted with ingenuity and indefatigable enthusiasm led them to conclude that facial palsy due to injury in the temporal bone should be treated by autoplastic nerve grafts. Other workers in the United States had previously reported intratemporal repair, notably Bunnell[2] in 1927 and Martin[3] in 1931, but the real stimulus for the developments that have made possible our present work on the facial nerve came from the collaboration of Ballance and Duel. My own interest in the subject began when in 1933 I saw in New York the results of Dr. Duel's operations and learned from him the technique that he had developed by that time.[4, 5]

The clinical success of Duel's facial nerve graft operations led to renewed interest in nerve grafting. Researches on nerve regeneration notably by Weiss,[6] by Loyal Davis[7, 8] and his associates in this country, and by the Oxford School in England[9-12] proved invaluable in dealing with the nerve injuries of World War II. These experimental studies have in their turn improved our understanding of facial nerve lesions.

Understanding of the basic facts of nerve and muscle pathology is essential when considering treatment of facial paralysis. The rigid facial canal may hinder regeneration but cannot alter the fundamental process of degeneration and regeneration. In the first place it is necessary to have a clear picture of the difference between degenerative and nondegenerative lesions. Unjustifiable operations, particularly for Bell's palsy, have undoubtedly been undertaken in the past from failure to make this important distinction. Rapid return of function sometimes claimed for decompression, occurring in a matter of days, cannot be due to regeneration of the nerve fibers.

Surgeons working on peripheral nerves recognize various degrees of nerve injury.[13] In transient block where conduction of the nerve impulse is temporarily impeded the paralysis clears up completely in a few days or weeks. Such a condition is responsible for the palsy that sometimes occurs a few days after a radical mastoid operation and clears up following removal of the packing in the cavity. In this transient block or "physiological interruption" there is no wasting of the muscles and the electrical reactions are unaltered. Varied degrees of ischemia may be responsible and the myelin sheath may disappear but there is no degeneration of the axons, that is, there is no reaction of degeneration.

Degenerative lesions are due either to complete division of the nerve elements when there is no possibility of spontaneous recovery or to interruption of the axons with preservation of the supporting structures. Wallerian degeneration occurs in both but because the Schwann cells and neurolemma tubes remain intact in the latter, regenerated axons can grow along the old paths so that spontaneous recovery is possible. There is no clinical difference between the two types of paralysis. Electrical testing demonstrates the reaction of degeneration in both and there is, at present, apart from exploration no certain means of distinguishing between the two lesions.

Here, therefore, is one problem: The prognosis for spontaneous recovery in the presence of a known degenerative lesion. There is, I think, unnecessary confusion in discussions of this subject because of the failure to distinguish facial paralysis due to injury or disease in the temporal bone from Bell's palsy. To my mind, the latter term should be applied only to those cases of spontaneous origin in which facial paralysis appears apart from other obvious neurological lesions as an isolated condition in the absence of recognizable ear disease. What I am about to say now refers, therefore, to facial paralysis of definite otological origin.

If facial paralysis is present immediately after an operation on the mastoid, whether a cortical or a radical operation, a severe injury must have been inflicted on the nerve. The nature of the injury—compression by detached bone, tearing of the nerve sheath or section possibly with loss of nerve substance—can be detected only by exploration. If proper care for the facial muscles is instituted at once (I shall refer to this later), operation is, however, not a matter of urgency. It is safe to wait until the modern methods for assessing nerve injuries and facilities for nerve surgery are both available. The lesion here is inevitably degenerative. Can we tell whether conditions are such that regeneration could take place spontaneously? That is, are the epineurium and the more intimate supporting struc-

tures of the nerve—the Schwann cells and the neurolemma tubes—
still intact? I believe this question to be academic here, important
as it is for the neurosurgeon dealing with peripheral nerve injuries.
Owing to the situation of the facial nerve in the narrow bony fal-
lopian canal, the effects of an injury that produces an immediate
paralysis are likely to persist and prevent myelination and matura-
tion of new axons. Exploration of the mastoid wound with exposure
of the facial nerve is essential. If the nerve is divided a nerve graft
will be necessary. I have never found simple section of the nerve
with the ends in apposition. Such an injury is, of course, a possi-
bility, but operation has in my experience revealed either compres-
sion or transfixion of the nerve by a fractured fragment of bone
with the nerve intact, a torn sheath with the nerve involved in
dense scar or a more gross loss of nerve substance of which the
original operator had been unaware.

Since a nerve graft may be necessary in these cases it may be
urged that operation should be undertaken at once. There are,
however, two good reasons for delay. In the first place infection
in the neighborhood of a graft or suture leads to fibrosis, both intra-
and extraneural, which prevents the regenerating axons reaching the
peripheral stump. Second, the outgrowth of Schwann cells from
the peripheral stump towards the center which plays the part of a
bridge by which the regenerating axons reach the periphery is most
vigorous between 15 to 25 days after division.[9] Delay, therefore,
for about three weeks will favor satisfactory regeneration.

If there is reliable evidence that the paralysis did not appear
until three or four days after the mastoid operation we know the
nerve cannot have been divided. I have in the past felt that this
certainty justified the surgeon in waiting until the electrical reac-
tions could be carried out. Packing in the mastoid cavity should,
of course, be removed. Then about 14 days after the onset of the
paralysis the electrical reactions are done when the presence or ab-
sence of the faradic response will tell us whether the lesion is a
transient block to conduction or a definite degeneration. This is
my usual teaching but I think we might today speculate on the con-
ditions that give rise to degeneration and question whether imme-
diate decompression might prevent the reaction of degeneration tak-
ing place. We are faced here with a paradox: A more serious
injury which has divided the facial nerve can well be left for two
or three weeks before operation until conditions for regeneration
are most favorable. But the problem case is the one where the
original injury has produced an interference with conduction which
of itself is temporary and recoverable, a condition that is sometimes

called neuropraxia, but where persistence of the compression in the rigid facial canal can produce changes which may lead to degeneration.

At this point I must digress to consider the results of regeneration. Perfect recovery of facial movements is never possible. Some degrees of synkinesia or failure of isolated action of individual muscles is inevitable in the face whenever recovery occurs after complete denervation, whether recovery is spontaneous or results from operation on the nerve—either nerve grafting or neurolysis. This diffuse distribution of new fibers which occurs after any total degenerative nerve lesion is particularly important in the face where the finer degrees of emotional expression depend on the isolated action of individual muscles or groupings of parts of muscles. Synkinesia may be minimal showing as slight retraction of the angle of the mouth on closing the eyes or increased narrowing of the palpebral fissure on the affected side, or the whole side of the face may move as a single functional unit which may be very disfiguring. Hence the importance of considering whether early decompression might prevent degeneration.

Denny Brown and Brenner[14, 15] have shown in some beautiful experimental work that the damaging effect of pressure on a nerve is due to occlusion of the blood vessels. This ischemia affects primarily the myelin sheath and has at first little if any influence on the Schwann cells and axis cylinders. Here is the temporary block to conduction I have already mentioned. But continued pressure produces other changes. Vascular stasis occurs in neighboring segments leading to edema above and below the site of compression. Not only is the process of repair in the compressed area impeded but a complete degenerative lesion involving the axis cylinders and the Schwann cells is finally produced.

We can now understand how reversible ischemia block, which may be present for lengthy periods in nerves of the limbs without leading to degeneration, will, by persisting in the rigid facial canal, produce increasing vascular stasis and edema leading to complete degeneration.

Decision for operation in these cases can only be based on grounds of probability. To explore in every case will certainly lead to the performance of unnecessary operations. To wait for unequivocal evidence of degeneration from the faradic reactions which cannot be obtained for 14 days would condemn some patients to the limited imperfect recovery which accompanies regeneration.

However, this consideration of the possibility of preventing degeneration by eliminating pressure in the early stages is largely academic as few patients are seen at this stage. We are more concerned with the patient whose facial paralysis has persisted for some time. Here the mode of onset and the condition of the middle ear and labyrinth will provide information on the type and site of the nerve injury. The time of onset, sudden and complete or gradual in development, is an important point in prognosis but the history can be relied upon only when the presence or absence of paralysis has been looked for and noted in the records. The onset of facial paralysis is easily missed in an unconscious patient with a bandaged head. This applies to patients with head injuries as well as after mastoid operations. I have on several occasions operated on patients whose facial paralysis was said to have first appeared 36 to 48 hours after operation and have found complete section of the nerve. If the tympanic membrane is intact the injury is likely to be in the vertical part of the nerve when repair can be effected without injuring the middle ear. Injury to the nerve at the bend below the external semicircular canal may have been accompanied by coincident damage to the external canal when a history of a labyrinthine "storm" at the onset and the presence of a "dead" labyrinth will be found.

But the chief problem in complete facial paralysis seen at an interval after onset is the character of the nerve lesion, degenerative or nondegenerative. The electrical reactions reinforced by electromyography will help in making this diagnosis. Unfortunately the reaction of degeneration gives no information about the possibility of regeneration of the nerve and reinnervation of the muscles; there may be good functional recovery while the electrical reactions are still abnormal even when tested under an anesthetic.

Recent advance in electrodiagnosis may help in these cases. The aim of muscle testing is to obtain a quantitative estimation of the proportion of normal to denervated muscle which the ordinary electrical reactions do not provide. Various stimulators have been designed which enable us to make comparable observations. The only one with which I have experience is the Ritchie Sneath[16] estimation of the strength-duration curve. The machine used gives stimuli of five durations. The voltage required to produce a threshold muscle twitch is determined for each duration of stimulus and the results are plotted graphically, giving the strength-duration curve. Denervated muscle gives a rapidly rising curve. As recovery occurs the form of the curve changes to that characteristic of normal muscle. The change in excitability indicative of recovery is noticeable between three and nine weeks before the first appearance of

voluntary power. This chart shows how the strength-duration curve may have prognostic significance. Electromyography is, however, of greater value, and I now employ this method of investigating the electrical changes in every case of facial paralysis where the faradic response is absent.

The development of valve amplifiers has facilitated the application of electrophysiology in the field of clinical medicine.[17, 18] By means of high grade valve amplifiers fluctuating potentials can be translated into sound on a loud-speaker or projected on the screen of a cathode ray oscilloscope. Needle electrodes are used which are inserted into the substance of the muscle to be tested so that samples are obtained of the electrical changes. It must be understood that a motor unit is the group of muscle fibers supplied by one motor nerve fiber. No electrical activity is detected under resting conditions in a normal muscle, but on voluntary contraction all the muscle fibers of the motor unit contract simultaneously producing an electrical discharge which, on amplification, gives a popping sound on the loud-speaker and a discrete diphasic wave on the screen. The discharges from different motor units are not synchronous so that when integrated they appear as irregular rumbles on the loud-speaker. For practical purposes we rely on the sounds emitted from the loud-speaker, as the detection of small differences in duration and frequency is easier by auditory than by visual methods. With increased voluntary effort the sound produced by these motor unit action potentials will be sustained. If this is not obtained we must suspect inactivity of a large proportion of the motor units. In complete axonal interruption of the facial nerve there is no recordable electrical activity at rest or at attempted movement for the first 12 days after injury. About the twelfth day a rhythmic activity called fibrillation occurs. This appears as repetitive potentials which have a ticking quality on the loud-speaker and small rhythmic spikes on the screen. Fibrillation is a manifestation of the autonomous life which is characteristic of denervated muscle. Fibrillation action potentials are therefore obtained as long as any contractile muscle fibers remain. If reinnervation occurs a muscle which has previously shown vigorous fibrillation becomes silent. We must, of course, remember that with needle electrodes we obtain only samples of the electrical changes from a restricted volume of muscle. Repeated observations may be necessary. Four or five insertions are made at each examination: in the forehead, in the orbicularis oculi, in the upper lip or angle of the mouth and the lower lip or chin. Total absence of normal motor unit action potentials and the presence of fibrillation action potentials indicate complete denervation. The

finding of both fibrillation and motor unit potentials is evidence of a partial or recovering lesion, and serial observations at intervals should be made to determine progress. Motor unit action potentials may precede signs of detectable functional recovery by as much as three months.

Two examples may illustrate the value of electromyography for prognosis.

REPORT OF CASES

Case 1.—W. K., a young man of 18 years had facial paralysis which had persisted for five months after a cortical mastoid operation. Reliable evidence of time of onset was not forthcoming but the paralysis was complete 36 hours after operation. The cavity was well healed, the tympanic membrane was intact and hearing was normal. There was no response to faradism but on electromyography a few motor units and fibrillation action potentials were observed in the orbicularis oculi and the muscles of the upper and lower lips. The finding of isolated motor units is not necessarily a guarantee of further reinnervation but if the nutrition and contractile power of the muscles are maintained by physiotherapy it is prudent to wait and repeat the electromyography at intervals. This patient was fitted with an intra-oral splint to support the muscles of the upper lip and given daily galvanism. Two months later vigorous motor units were observed though there was still no evidence of voluntary movement. Six months after the first electromyography the patient could close his eye completely and move the angle of the mouth voluntarily and in smiling. Synkinesia was slight and the result was as satisfactory as could be expected after any nerve operation.

Case 2.—A. H., a young man of 19 years had facial palsy of gradual onset with acute mastoiditis for which an operation was performed. Four months later there was no sign of recovery and the complete reaction of degeneration was present. Electromyography gave vigorous fibrillation in the orbicularis oris and the chin muscles and distant motor units and fibrillation in the orbicularis oculi. Physiotherapy and support for facial muscles did not lead to any improvement. Repeated electromyography during three months showed no increase in motor units but persistence of fibrillation. Exploration showed the nerve involved in dense scar in its vertical course with a lateral neuroma. There were few nerve bundles visible when a trial incision was made into the neuroma. The neuroma was therefore excised and a fresh nerve graft inserted. Examination of the neuroma showed intraneural fibrosis with scanty nerve fibers.

Absence of fibrillation action potentials in late cases of facial paralysis (provided the face is not cold) is evidence of loss of contractile tissue from atrophy and like the negative galvanic response is a contraindication to any operation on the nerve. I see many patients with a partial recovery of facial movements who have been sent for an opinion as to the advisability of nerve grafting. The presence of some motor units and no fibrillation in such cases shows that reinnervation has occurred in some muscle fibers and that the rest are hopelessly fibrotic.

I am not claiming that electromyography should replace the ordinary electrical reactions in facial nerve surgery but that the combination of electrical testing and electromyography affords a more accurate means of assessing the state of nerve and muscles. Normal innervation in a paralysed muscle, partial or complete denervation and minimal reinnervation before any observable return of voluntary or emotional movement can all be shown by this method of examining the spontaneous electrical activity of the facial muscles.[18]

Nor is electromyography always easy to interpret. For this reason I never do the testing myself but rely on the co-operation of one of my physical medicine or research worker colleagues, but I am always present at the examination as through this work I have learned a great deal of the natural history of nerve injuries.

Our understanding of the nature and degrees of nerve injury has developed since the time of Ballance and Duel. We know now that some mass movements in the face and contractures depending on this synkinesia are inevitable with regeneration, and that early operation in the presence of degeneration cannot prevent their development. The rapid, complete and perfect recoveries claimed for early operation were the result of operating on patients with ischemia block whose nerve fibers had never degenerated. I know that many but not all of these operations were undertaken in the presence of negative faradic responses which were held to indicate the reaction of degeneration. I do not think enough attention has been given to the possibility that the ischemia brought about by pressure may produce a mixed lesion, some of the axons undergoing degeneration and giving the corresponding electrical reactions and others being in a condition of reversible ischemia block, capable of recovering without degeneration. Electromyography combined with electrical testing has helped us to elucidate this partial lesion.

Much attention has been paid to the mass movements which mar the standard of recovery after regeneration. I think the feature which has equal importance in producing asymmetry is the

wasting of individual muscles or parts of muscles.[19] Denervated muscles become flaccid and undergo progressive atrophy which is most rapid in the earlier stages. Wasting is also more rapid during growth, that is, in infants and young children. As the contractile tissue wastes there is an increase in connective tissue, which finally obliterates the terminal Schwann tubes of the degenerated nerves and the motor end-plates. Before this stage is reached the changes are reversible if reinnervation occurs, but the greater the atrophy and interstitial fibrosis the worse the ultimate recovery. Muscle atrophy and the resulting fibrotic changes are soon apparent in the thin musculature of the face where there is no definite muscle fascia surrounding the individual muscles. These are inserted into subcutaneous tissue or skin without the intervention of any subcutaneous fascia. When denervation has persisted for any length of time the fibrotic sheet which replaces the individual muscles cannot be made to contract even if continuity of the nerve could be restored. The importance of physical treatment of the facial muscles in every patient with seventh nerve palsy cannot be overestimated. Reinnervation of the facial muscles even without operation is likely to occur in the majority of cases but because of the wasting and fibrosis that accompany denervation the quality of the recovery depends directly on maintaining the contractile power of the muscles. The best surgical technique which restores conduction of the nerve impulse by neurolysis or through a nerve graft is useless if the facial muscles have been allowed to stretch and atrophy before the operation or while nerve regeneration is taking place.

There is recent experimental and clinical evidence that atrophy and fibrosis in denervated peripheral muscles can be retarded if muscle function be maintained.[20] Muscle contraction is essential and this can be provided only by galvanic stimulation.[13] Galvanism does not prevent the atrophy that immediately follows denervation but it retards its progress. It was believed at one time that electrical treatment favored the production of contractures in facial paralysis by overstimulation. We know now there are two kinds of contracture: those which are a manifestation of synkinesia and in some degree inevitable where recovery is by regeneration and those due to a true fibrous contraction of newly formed connective tissue, the formation of which can be retarded and diminished by galvanic stimulation. It is, of course, understood that electrical treatment has no effect on the paralysed nerve but acts only by limiting muscle wasting and fibrosis during denervation.

It is difficult to prevent stretching of the facial muscles during denervation. Gravity and the pull of the muscles of the opposite

side, acting continuously in the play of facial expression as well as by voluntary movement have to be counteracted. The customary hook in the angle of the mouth attached to the ear defeats its object, stretching not only the orbicularis oris when the sound side contracts but the levator and depressor muscles inserted into it. Strapping with adhesive plaster or adhesive cellophane will keep the muscles relaxed. Support of the orbicularis oculi in the lower lid, wasting and stretching of which is responsible for persisting epiphora, can be prevented only by keeping the eye closed with cellophane strapping applied so as to raise the lower lid keeping the lacrimal punctum in contact with the globe of the eye. Strapping interferes with the more important electrical treatment and if taken off daily for this leads to soreness of the skin so that treatment may have to be suspended. For the past four or five years I have been using an intraoral splint[21] fitted to natural teeth or dentures so adjusted that the elevators of the upper lip are supported, but I am not satisfied with any of the means yet devised for preventing stretching of the facial muscles. If the plastic surgeons could insert temporary fascial strips superficial to the muscles without interfering with reinnervation these would be more effective than any mechanical splinting. Co-operation between the otologist and the plastic surgeon is essential at every stage of the treatment of facial paralysis. Apart from the plastic operations which are the only available treatment for the disfigurement associated with complete fibrous atrophy, the appearance can often be improved by small plastic operations such as tarsorrhaphy when wasting limits recovery following nerve surgery. Sometimes a fascial strip operation in addition to masking the deformity allows partially recovered muscles to work at better advantage so that movement hitherto ineffective becomes apparent.

I would like now to refer to one or two details of operative technique where experience and advances in peripheral nerve surgery have led me to modify the operation as originally devised by Duel. Operations on the facial nerve are tedious more than difficult. Experience in mastoid surgery and practice on the temporal bone are, of course, essential. Previous operation or disease may have altered or removed the normal landmarks. Sometimes the opening of the eustachian tube is the first recognizable feature of the operation area. After gunshot wounds and in some cases of operative injury, especially in infants when the mastoid process is not developed, the trunk of the nerve may have been severed after its exit from the skull. In such cases the lower end of the nerve can be found by isolating the cervical branch and following this upwards to the trunk. There is no danger of a parotid fistula if the

parotid duct is patent.   Persistent bleeding from the stylomastoid artery in the bone tells one the nerve is near, if it is necessary to expose the nerve directly in the bone.   Hot saline and suction or fibrin foam are used to control hemorrhage but one must remember later in the operation not to use suction when the nerve graft is in place.   I once lost a fine graft when an enthusiastic assistant tried to tidy up the cavity.

When the site of injury is reached excessive scar tissue may surround the damaged nerve so that it is difficult to tell whether there is complete section, tearing of the sheath or an intact sheath with adherent scar.   The binocular operating microscope is useful here.   Nerve bundles may be recognized in the severed ends.   It is important to decide whether the nerve is completely or partially severed.   When the division is complete the central end must be looked for.   Sometimes this is involved in granulation tissue at the site of the geniculate ganglion; the operating microscope is useful for finding whether there are nerve bundles present in the stump. Adequate resection of the scarred ends, especially the distal stump, if possible until obvious glistening bundles are seen, is important for ensuring successful regeneration.   This is difficult to judge in the small facial nerve but some poor results are, I am sure, due to neglect of this detail of technique.   Both the graft and the peripheral end must be handled gently, as successful regeneration depends on the active co-operation of the living Schwann cells.   A razor blade is used for cutting nerves where possible as this inflicts minimal injury.   Where a razor blade cannot be employed in the bone, fine iridectomy scissors are used.   As grafts tend to shrink and shorten it is important to take a graft longer than the gap between the two ends of the nerve.   I believe that the limited recovery in some of my earlier graft operations was due to failure to allow for shrinkage. If the apposition of graft and the ends is not maintained the regenerating axons escape from the junction and form a neuroma.

If the inner wall of the fallopian canal is still present (sometimes it has been completely obliterated at the previous operation) it supplies a rigid channel in which the graft can be placed.   In any case I always use the so-called fibrin suture.[22]   This does away with the need for sutures thus minimizing scarring at the nerve junctions. A firm clot which glues the ends of the nerve and the graft can be made from fibrinogen and human thombrin.   No further protection of the nerve is necessary.   This fibrin clot is more satisfactory than the patient's own blood clot as a clot of whole blood is weak and messy and rapidly contracts to give a spongy tangle of fibrin

fibers and red cells while the fibrin clot remains for some time as a firm, transparent jelly.

When the nerve is intact pressure outside should be relieved by removing detached fragments of bone or adherent scar. This uncovering of the nerve should be done well above and below the site of injury. Complete decompression by slitting the sheath is not always necessary. Removing the outer wall of the fallopian canal is like taking away the crutches from a patient with crutch palsy. If the sheath is torn and there is adherent scar, incision of the sheath may be necessary. In active suppuration openings of the sheath should be avoided as it predisposes to intraneural fibrosis. This is particularly important in chronic suppurative otitis media with pre-operative facial paralysis.

Sulfonamide powders should not be used in these operations or indeed in any operation where the facial nerve is likely to be exposed as they interfere with regeneration.

The after-treatment of the wound is most important. No attempt should be made to remove blood clot or secretions except from the outer part of the meatus for the first few days. After ten days the cavity should be found smooth and shining. If granulations become excessive it is better to let them form rather than to endanger the nerve by cauterization or curetting. One of my cases suffered from curetting when I was absent at the war. I must confess that I am not satisfied with the methods at present available for treating the mastoid cavity after a nerve graft and would like to know the experience of others on this problem. A classical radical mastoid cannot always be expected, nor is a temporal muscle graft satisfactory protection for a nerve graft. On two occasions I have had to reopen mastoid wounds where the surgeon had thought he could protect an exposed facial nerve by a temporal muscle graft and found dense scar involving the nerve sheath. It is possible that the alginate dressings which some otologists are now using in the fenestration operation will facilitate healing but before they can be tried experimental investigations on their effect on nerve junctions are necessary.[23]

This problem of the mastoid wound does not arise when the nerve injury has been restricted to the vertical course when decompression or nerve grafting can be carried out without damaging the middle ear, if this is intact. Here the wound can be sutured after the fibrin clot has formed.

I do not need to say much about Bell's palsy. Treatment of a condition the etiology and pathology of which are in doubt can only

be empirical. More than one pathological process is probably responsible for facial palsy occurring apart from injury or disease of the temporal bone. Merwarth[24] has separated a small group due to intrafallopian hemorrhage in patients with malignant hypertension, analogous to the conjunctional and small retinal hemorrhages that occur in these patients. I have seen two that could be ascribed to this cause. The occurrence of cases in groups or small epidemics suggests an infective origin probably from a neurotropic virus.[25] I came across Bell's palsy more often, especially in children, during the 1947 epidemic of anterior poliomyelitis in England. Few cases of Bell's palsy come to autopsy. Those reported suggest that the lesion may be in part a poliomyelitis and in part a neuritis.[26] There is no doubt that some cases have changes in the cerebrospinal fluid during the period of onset.

The operative findings on decompressing the facial nerve in Bell's palsy have been various. Some surgeons have found the nerve swollen and edematous. Cawthorne[27] and others have described a constriction at the stylomastoid foramen. Kettel[28] describes necrosis without inflammatory signs in the mastoid and postulates a lesion of the vasa nervorum. I have found scar tissue, a constriction at the foramen, but more often no abnormality. The results of operation in the presence of a degenerative lesion have varied without relation to the operative findings. Because we cannot be certain whether the particular lesion is central to the middle ear (testing for taste and for suppression of tears is seldom of practical value in my experience) I have been led to doubt the value of decompression operations in Bell's palsy.

Ballance and Duel advocated nerve grafting rather than nerve anastomosis in every case of nerve section because it restored emotional as well as purposive movements. They did not allow for the possibility that the central end might not be accessible. There is still a place for facial-hypoglossal anastomosis, particularly when the middle ear is intact, for example after removal of an acoustic neuroma or for persisting facial palsy after fracture of the base of the skull. Symmetry at rest may be restored and purposive movements to order, even some voluntary smiling, but spontaneous emotional movements do not occur. The obvious disfigurement that thus appears as the face drops on laughing and smiling can be minimized by fascial slings. Spontaneous emotional movements are always present when a nerve graft restores the normal central pathway with the periphery but the finer shades of emotional expression are limited by the degree of synkinesia.

CONCLUSIONS

This brings me to summarize my views on the present position of facial nerve surgery. We have now, at any rate theoretically, developed an operative technique that minimizes formation of scar tissue thus facilitating satisfactory nerve regeneration. Progress now is needed in three directions: First, we should consider whether we can improve the after-treatment of the radical mastoid cavity after nerve grafting so that early epithelialization occurs without hampering regeneration of the nerve. In the second place there is need of better treatment of the denervated facial muscles to prevent wasting, stretching, and the resulting weakness. Here we look to the physiotherapist and the plastic surgeon with a warning to the physiotherapist not to allow attempts at movement until voluntary movement is possible. Finally and most important there is a need for more precise indications for operation: In early cases can degeneration be prevented where, without interference, it is inevitable? In late cases where degeneration has already occurred what is the optimum time for exploration? We know from experimental studies on divided nerves that with delay in suture there is progressive shrinkage of the Schwann tubes which results in defective myelinization. There is also progressive atrophy in the end organs which may prevent new nerve fibers from entering the old nerve plates. In peripheral nerves the outlook after one year's denervation is very poor; how much more in the delicate facial muscles where muscle fibers are more easily overcome by interstitial fibrosis. Hence the importance of an accurate history of onset and the correct interpretation of diagnostic aids to enable us to obtain as clear a picture as possible of the condition at the site of injury.

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Scientific Papers of the American Otological Society

<center>LXV</center>

<center>PRESIDENTIAL ADDRESS</center>

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<center>New York City, N. Y.</center>

We are living during great times. We are singularly blest by being in the medical profession and especially in otology during these great times. Are we using our maximum mental and physical efforts to the best advantage?

The American Otological Society has among its members the majority of proven leaders in American otology. Because our eminent membership places us in a position of leadership, there are responsibilities which the American Otological Society must accept. Our responsibility is to maintain that which is good in the traditional and disseminate knowledge of that which seems to be good in the innovations. Another responsibility which we must accept to ensure a healthy future for our society is the recognition and encouragement of young, thinking workers. Still another responsibility is the broadening of interest and opportunity in the fields of science which can contribute to otology. Advance in knowledge can best be made through our co-operative effort.

As otologists, it is desirable for us to encourage other scientists to work with us. No otologist can possess all the important knowledge necessary for our development.

The American Otological Society is rightly considered a senior society. As such we are expected to give counsel and encouragement to younger men. Such encouragement to the younger men should be valuable, available and active. Perhaps our most valuable asset is the mature judgment which can only be acquired as the result of

many years of thoughtful experience. This last sentiment is a heritage from my very dear friend, the late Dr. John B. Rae.

Each member of our American Otological Society has, at some time, made his contribution to otology. Such contributions are among our requirements for membership. Each member can therefore feel a justified pride in belonging to at least one society in our modern world which holds "giving" instead of "getting" as one of its membership requirements. The contributions which each member has made are for the benefit of every individual in this world. Our gift is not made with a label designating which color, race or creed shall receive it. And no comment is made in the distribution of our gift which would make us conscious of the existence of such divisions.

This society also places honesty, fairness, mutual confidence and ethical conduct high in its required qualifications for desirable members.

The citation of these attributes is not to be dismissed as trite; these qualifications in potential members are carefully considered. Modesty and humility in candidates are preferred to beating of personal drums and beating of personal gums. Such discordant sounds emanating from candidates fall on the ears of men who suffer from an unsympathetic perceptive deafness.

Every person who meets our scientific, mental, moral and ethical standards is cordially welcomed to our membership and to our meetings. We welcome you to this, our eighty-second, annual meeting, at the Biltmore Hotel in New York City.

A loved and famous New Yorker made one phrase popular with those who seek the truth: "Let's look at the record," said Al Smith. A critical time has arrived when we should examine our own record of the past, consider our present and plan for our future intelligently.

A few years ago (about 400 B. C.), Hippocrates formulated a moral code for medicine. This code is accepted under oath by those entering the practice of medicine today. Medicine as a whole has lived by this code in a surprisingly faithful manner. Almost universally medical men agree on what is right and wrong in medical conduct. There is no force which compels doctors of medicine to live by these moral laws. The only enforcement agent is the desired good opinion of medical brotherhood. When we look at our record, we can be proud of our accomplishment. There is also a universal religious code of right and wrong which in its essentials is common to all religions. Medical men have joined, with a minimum of

prejudice, in following not only their own medical codes but also deep down in their thoughts men of medicine hold to a universal religion which is so simple and elemental that it approaches eternal truth. Many serious shocks to our brothers in medicine have not perceptably changed either our ideal or our ideals. Modestly we can claim that what we have accomplished in right living should serve as an example. In these days, we may now be truly thankful that we have always adhered so rigidly to our principles. On the basis of our past record, we can be sure of our courage and faith in the future. Our leadership in medicine and in the world order must be maintained. The crucifixion of medicine in England must surely be followed by a resurrection. Our own crucifixion may be pending.

Where is otology's place in medicine? Otology as a specialty is of recent development. Our specialty had its beginning well within the memory of some otologists here present. We have, therefore, little in tradition to guide us. Lacking traditional guidance, we must get our bearings and, like good navigators, plot our future course. The ability to do a job better than the other fellow is and always will be the only excuse for any specialty. If we are to maintain our usefulness, we must continue to build a better otological mouse-trap.

Time, as would be expected, has brought changes. The first honest seekers after truth prospered and their prosperity was noted by others who sought reward without the necessary labor. Self-styled surgical specialists in all fields whose training and ability were far below that of the average surgeon collected the spoils on the assets of personality plus the specialist aura, which was assumed but not earned. Fortunately for the patient, this condition has been largely remedied through the laudable efforts of the American Specialty Examining Boards. The qualifying boards are youngsters, and like some youngsters they occasionally deserve, and get, a spanking. The good which the Boards have done as a whole far outweighs the harm done by some more recent boards who seem to have developed a superiority complex. To be a good surgical specialist, one must be a good surgeon. A good, broad, medical training is and will continue to be a prerequisite for both. The over-trained superman is rare and of questionable value.

Medical progress is apparently stimulated by the challenge of obstacles. In our early days, anatomical knowledge was gained through the efforts of zealous students who risked imprisonment

and even their own lives in order to learn about the then unknown human anatomy.

Our present advance in the prevention and cure of disease in the human could not have been accomplished without the knowledge gained through experimentation on animals. For many years, doctors have had to waste time and money to combat the efforts of those well-meaning but misguided people who are constantly attempting to obstruct medical advance.

A world-shattering economic depression and a couple of world wars have curtailed the customary research activities in our time. Perhaps the only good that has resulted from any war is the increased medical knowledge gained which can be used in the care of the peacetime sick.

A "Humpty-Dumpty" governmental economic and social program has been especially burdening to the medical profession. The physician belongs to one of the few groups for which economic security against sickness and old age is nigh impossible. The physician has resented and fought against biased legislation but at the same time he has continued to do his job conscientiously. In consequence, he has improved medical care to a point which is unequaled in any other country. Medical science will continue to advance as it has in the past in spite of adversities.

Otolaryngologists and, more particularly, otologists have had the added burden caused by a diminished surgical practice. This diminution in surgery has been brought about largely through our own efforts in preventive medicine. The decline has been accelerated by the sulfa drugs and antibiotics, but a marked decrease in the incidence of surgery started two years before the use of sulfa drugs in this country. Notwithstanding our diminished revenue, there has been a justified feeling of satisfaction on our part because we are proud of our accomplishment. What other group of humans considers a drop in their income as a success?

During the affluent days of a somewhat avaricious surgical era, the less spectacular problems of impaired hearing received scant attention. A little in accomplishment was recorded by a few courageous souls, but the average otological surgeon was ignorant of facts related to hearing impairment and uninterested in learning. As much as some of us may hate to admit it, perhaps the greatest force which turned some specialists' minds to the problems of patients with impaired hearing was the spectacular fenestration operation. Whatever the cause, there is now a healthy curiosity regarding the organ of hearing.

Our researches have progressed slowly because there has been a minimum of financial aid. All our appeals for funds seem to fall on the congenitally deaf holders of purse strings. The handicap of a person with impaired hearing does not stir a strong emotional response in persons who are not similarly handicapped. Even the Veterans' Bureau declares rather callously that when the veteran is furnished with a hearing aid, he is one hundred per cent rehabilitated and refuses to pay the veteran's justified compensation.

It seems to annoy some people when a person with whom they are talking is unable to hear their low-audibility mumbling. The person with impaired hearing offers no visual emotional appeal, perhaps on the contrary, people are repulsed by supposed stupidity.

The fear of cancer, pity for the deformed, the exaltation of someone furnishing the means to return sight to the helpless blind, agents who free the tubercular from their confining beds, the cruel statistical stab of the cardiac; all these emotions are rightly or wrongly aroused to procure funds for organizations devoted to the care of such sufferers. We have been forced to overcome the handicap of the proportionately small funds available for those aural cripples who lack emotional appeal.

We have great difficulty in obtaining the services of the highest type of scientists with mutual interests who work in other scientific research. Although they should be allied with us in our studies, in salary competition for their services we are hopelessly outclassed by industry. In industry, these top scientists may be compensated in proportion to the income produced for their company by their discoveries. Unfortunately, the results of our research do not increase our income. Although we can only afford a small money compensation, we can promise a reward in sharing our exaltation when we find something which will give people an increased living capacity, utility and pleasure. "Idealistic. Not practical!" would be the expected comment by the contemptuous sophists of today. But the fact remains that this same idealism adds a zest to living which the dollar motive has not produced. Overcoming these and many other difficulties, otology has advanced, and some of our conquests, in retrospect, are little short of miraculous.

Meningitis was, until recently, a dreaded word synonymous with death. Today, death from meningitis is a rare occurrence.

Purulent acute otitis media and its sequential mastoiditis have been almost completely eliminated today. And again I wish to emphasize the diminution started before sulfa.

Complications of ear diseases, such as brain abscess, cerebellar abscess, sigmoid sinus thrombosis, and petrositis are rarely seen today. The annals of our society have been enriched with the contributions on sinus thrombosis. The Boston ligationists and the New York resectionists furnished verbal fireworks for the enlivenment of many of our not-so-ancient meetings.   The good old days of petrositis with its tapped reservoir of pus rolling forth like the contents of a cream puff are a lingering memory.   Surgery for the cure of these infections bids fair to be among the lost arts and we surgeons should feel most discouraged.   Strangely enough, we feel elated over our accomplishments.   Having developed our abilities by conquering these very difficult problems, we feel stimulated and encouraged to look for more and more demanding fields for conquest.   Impaired hearing, tinnitus and disturbed equilibrium seem sufficiently formidable for a start.   One type of impaired hearing is correctable through surgery, but this surgery must be considerably improved and simplified before the surgically accomplished cure of deafness can be considered as generally accepted.   Operations for the cure of tinnitus and disturbed equilibrium have also been performed and the published results sound hopeful.   However, the fickle desertion of surgery as our provider in our not-too-distant past makes us a little hesitant in accepting and depending on its future support, without reservations. We had ample warning of surgery's intended infidelity. We must learn from experience.   During the past era of surgery, we felt ourselves a little too superior as ultraspecialists. A little humility without servility may adjust our thinking on a more useful basis. The former superaristocrats who posed as left ear specialists will have to broaden their base and reduce the height of their personally created pedestal.   They may have to revert to the ancient and honorable title of "Eye, Ear, Nose and Throat Men" in order to best serve their supporting public.   I am inclined toward the belief that the public might be benefited by such a homely tendency.   But we must never forget to maintain our high ideals as an emblem of hope and inspiration.   This highly scientific but humane society must continue to encourage otological progress and thus continue to furnish such an emblem.

The future is always bright for selfless people.   It may even be bright for selfless otologists.   Perhaps our greatest faults have been an assumed supercilious scientific superiority and medical isolationism. If these have been our faults, they are correctable.   We must graciously admit that we do not know everything about all the subjects presented at our meetings.   We must come to these meetings

humbly admitting our lack of total knowledge and not be ashamed to learn. To end our isolationism, we must request our medical friends in all fields to enlighten us with their knowledge of related scientific subjects. Again I mention Dr. John B. Rae because he had the vision to establish a precedent by inviting a physicist to appear before this society. The implications of his innovation have not been fully grasped. Many of the scientists working on allied projects have stimulating information which is useful in our own investigations. There are many scientists outside the medical circles who could and would work with us effectively in solving some of our problems which to us, with our limited knowledge, are insoluble. Even in our own medical circles, there are many doctors in other branches of medicine who would willingly work with us if they were properly approached. I am afraid we have been rather myopic in our tight little specialty field. It is time we broadened our vision to include biologists, chemists, pharmacologists and many others in a group effort. The pathology, anatomy and embryology of the ear has received major attention in the past; now we want to know about the living action and disturbed function of the organ of balance and hearing. Our present knowledge is shockingly meager.

Our main efforts of the past were devoted to curing infections of the ear. Infections as assets have depreciated in value and now our efforts for the future must be invested in something else. What better place to start anew than with the study of physiology, chemistry, and electronics in order to better understand the functions of our own special piece of the human anatomy. We cannot do this job alone and neither can anyone else do the job without us. But a co-ordinated effort will be successful. Brother medicos may steal our tonsil operations, our myringotomies, our endoscopies, our plastics, and almost our shirts, but few have the special knowledge needed for courage to poke their untutored noses into the organ of hearing. We are still in possession of the special knowledge one needs to intelligently deal with aural problems. We are glad to offer our special knowledge as our contribution in a joint effort to cure more human otological ills.

In our future studies we must adjust our thinking to simulate the mental processes of scientists of which the nuclear physicists are an example. We have thought in terms of tangibles; we must now think in terms of the abstract. The structures and forces with which we are working are of an incomprehensible delicacy. Our past efforts to understand the action of the ear have been crude compared to the precise methods used in some other fields. In the

vestibular apparatus we are dealing with measurements, weights, osmotic balance, chemical variations, stimulae, impulses, wave frequencies, electrical currents, and physical phenomena which are much too small for mental visualization. In the past we have considered ourselves quite erudite because of such accomplishments as learning the microscopic postmortem appearance of the inner ear structures. We are just realizing that while this effort has been laudable, we are still a long way from complete understanding of the normal function of these structures in life. We must realize that the tissues of the inner ear undergo postmortem changes and changes due to the chemicals used in their preparation for examination, which alter their appearance to an undetermined degree. It is only in recent years that we have been able to see the tissues of the inner ear in vivo with a low power microscope. And even in this instance we have probably disrupted the normal order of things by our forced entrance necessary for exposure. The highest magnification we are able to use is insufficient to visualize the action of those structures involved in hearing and equilibrium. Our attempts to understand the ear have almost been in the class of wishful thinking. Perhaps we can borrow an illustration from atom research to support our argument. We know that some of the information gained in atomic research ·is applicable in otological research but to what degree we will be aided is not known.

These statements are for the kindergarten group in the study of nuclear fission. An energy-measuring unit is an erg. An erg is the kinetic energy of a mass of two grams moving one centimeter a second. Atomic processes are measured in micro-ergs or micro-micro-ergs. A micro-erg is one millionth of an erg. Here is a force too small for human perception, yet it is a measurement used in the most destructive agent yet known. The atomic energy concealed in one gram of uranium is equal to nineteen tons of T.N.T. In the atom there is a nucleus which is surrounded by an atomic wall. The space between the material of the nucleus and the wall is a vacuum. If the fluid substance of the atom or nuclear fluid which is dispersed through space could be collected to form a continuous material, one cubic centimeter of it would weigh two hundred and forty million tons. I think you will agree that such a condition is too great for the mind to grasp. We also find in nuclear physics that various electrically charged particles are shot through space at the incredible speed of over ten thousand kilometers a second. In addition to the charged particles there is an energy propagated as electromagnetic radiation manifested by heat, light, x-rays, gamma rays and cosmic

rays. Which one of these unbelievable but real forces holds the secret stimulus which is involved in auditory microphonics or the action waves of the auditory nerve? To solve our problems we seek co-operation with others in order to pool our knowledge.

These things are real; they are not idle vaporings of the undisciplined daydreamer. Such facts must blast their way into our serious thoughts and be recognized. Less than one hundred years ago the methods we use today were new and exciting, but they can become uninspiring, repetitious, inadequate and antiquated. We have been mentally coasting on the full years which the alert minds of our pioneer predecessors produced. We have experienced the discouragement of a period of troubles. We can still live comfortably by using the proven methods of our traditional otolaryngology. However, we must be able to gain the necessary impetus for advance from the rapidly moving events around us in order to visualize and be stimulated by a newer inspiring otology. Some of the workers on atomic projects, with whom we have talked, commented on the opportunities they could see ahead of us in otological research and enviously remarked how happy we should be. They said they had been using their scientific brains to produce agents of human destruction while we otologists use our minds to enhance human living. We medical men in otology should be exhilarated. We are among the few men of today who are free, truly free. We must use our brains, energy and imagination in a campaign of action if we are to remain truly free.

Our campaign should include:

1. The enlistment of the best minds for a co-operative otological research.

2. The enlistment of funds to be distributed through our own board of trustees for our research fund.

3. Encouragement of research by an award such as has been proposed by Dr. Bernard J. McMahon, my predecessor in this office. Such an award will be presented for the first time this year.

4. The stimulation and aid for those young workers who show ability by granting fellowships.

5. By keeping our program reserved for the best in scientific and applied otology.

121 East Sixtieth Street.

LXVI

THE PRELIMINARY EDUCATION OF THE PHYSICIAN

Harris P. Mosher, M.D.

Marblehead, Mass.

When the Council kindly asked me to be Guest of Honor at this meeting of the American Otological Society, under Doctor Jones' chairmanship, I felt the honor deeply, and sincerely thank the Society for it.

My subject is "The Preliminary Education of the Physician." The paper begins by reviewing my college course, especially what I now consider its faults and omissions, and then takes up the premedical education of the medical student. It is a very personal paper.

I realize that for both you and me, much of what I have to say is "water over the dam," but I am consciously talking over your heads to a younger group and, perhaps, through some of you, to a son who is coming along.

MY COLLEGE COURSE

The elective system was in full swing during my college days. It was new, and soon became the fetish of all universities. The student was unguided and made his choice of courses and subjects with what little maturity he was fortunate to have. Things, as you know, are different now.

In my Arts course, I survived all the courses in English composition up to and through the final one. I have forgotten most of the so-called rules I then learned. This probably matters little, as the only rule today seems to be to write, and write, regardless. In the final course given by Professor A. S. Hill (Ass Hill, the students called him, inspired by his initials), I found myself with a man on my left who later became Professor of English in Chicago, and on my right, a poet who had already given great promise, but soon died young. Next to the poet was a student who became a Justice of the Supreme Court of Massachusetts. Naturally, I was unhappy in the

Looking back, I feel that there are two priceless things which every college student should learn: To write clear, simple and un-

midst of so much potential ability, but I struggled through the course.

varnished English, and, as the common phrase has it, to be able "to talk on his feet," and not have to be of Irish descent to do it. I have been thanked more by house officers for the opportunity to practice along this line than for anything else I was able to do for them.

My greatest regret about my college course is that I took so little history. I came out of The Boston Latin School, the oldest public school in the country, with a parrot knowledge of Greek and Roman history. This soon faded out, as it was only factual history. My lack of knowledge of interpretative history has been a life-long humiliation. It is a platitude, but a true one, that the past makes the present. You cannot even live down your own past. Try it, and see!

Here, allow me to quote Whitehead. He was an Englishman, a mathematician, a physicist and, finally, a Professor of Philosophy at Harvard for some ten years. He has just died. Whitehead says, in one of his aphorisms: "The University should impart information imaginatively. Imaginative consideration transforms knowledge. A fact is no longer a bare fact; it is invested with all its possibilities."* History taught this way is what I call interpretative history.

A second regret which I have about my college course is that I graduated without a conversational knowledge of either French or German. I feel that every college should have a dormitory where the students living in it hear (steadily, for four years) one foreign language, and are taught to speak it. Repeatedly, I have been embarrassed by inability to talk with foreign visitors beyond a few phrases of greeting.

PREMEDICAL EDUCATION

My objection to the premedical education which is required today is that it can shorten the liberal arts course too much. My premedical education would never get me into a medical school today. As I knew I was to go into medicine, I decided to get as much general knowledge as I could since it was to last the rest of my life. I have, as you have, since leaving medical school, literally spent my days and nights at medicine, and medicine requires more overtime than any other profession.

By the way, I have no clear idea now why I went into medicine. I wonder how many of you know why you chose medicine as a career. Few doctors go into medicine simply to do good. No doctor,

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*"The Wit and Wisdom of Whitehead", Boston, The Beacon Press, 1947, p. 96.

except one of the brow-stroking type, whom I despise, likes to be called a "do good-er." It may have been the mechanical side of medicine which attracted me. My father was a civil engineer and a bit of an inventor. Having, as I just said, spent my days and nights for so many years in medicine, I have neglected some cultural subjects, and I am now, in what time is left, trying to catch up on them. I always hate to use the word "culture," it sounds so snobbish, but no other word takes its place, and it is supposed to be the favorite word of the "Proper Bostonian," and I should be proud to be called a "Proper Bostonian."

Now that I am looking backward, I wonder if giving a hundred per cent of my time to medicine over the years was not a little too much. Five or ten per cent less, used judicially and systematically, would have prevented my present scramble to catch up on cultural subjects, and have made me less lop-sided.

Getting back to the subject of the paper, namely, premedical education, I miss not having taken in college, biology, and comparative anatomy. I have had to get a smattering of these subjects by myself, in order to broaden my teaching.

It is easy in premedical education to overstress physics and chemistry. There are in medicine, as in athletics, "naturals." In medicine, they are the men whose personality makes them quickly successful physicians. They become the mainstay of every family they enter, a mainstay mentally, morally and physically. By comparison, the cloistered scientist is a mere laboratory animal.

The great objection to giving four years entirely to the liberal arts is the time and expense involved. This can be a fatal objection. Government scholarships are in the air, and could help. Of late, college presidents have begun to edge up to them.

THE SUPPOSED ADVANTAGE OF A COLLEGE DEGREE

As entrance to a medical school has long required a college degree, the men in medicine who did have it were of my time or near it. They were haunted day and night by the tragedy of what they supposed they had lost. It is notorious that many holders of a college degree practically missed education. Probably, they got what they aimed for: social acquaintances on which to capitalize through life, or athletic fame which later was useful in the same way. They went through life on their first names, or nicknames, but their names are seldom found in "Who's Who!"

I have the fullest admiration for those who came up to medicine the hard way. Their life stories would put most of us to shame. There is a "stalking grandeur" about most of them. But I have

seen more than one physician make up for the lack of a college education by planned reading and study. This is now called "continuing education" or "lifelong learning," and I might add that it is the only true form of education. Education should end only at the grave. Think what we might learn if it could be continued beyond!

I hope I have said a word of comfort to those lonely souls of my time who continue to mourn the loss of a college education. This seems to be a wound which seldom heals. I should be very proud if any word of mine could start the healing of such a wound.

THE MEDICAL SCHOOL CURRICULUM

One begins to hear a word of warning about the overloading of the medical school curriculum due to the rapid advances in medicine. The modest size and equipment of the doctor's bag of my day (a sort of mongrel Boston bag) has become a suitcase, and soon will become a trunk.

Today it is a terrifying thing to start the practice of medicine. It would almost seem that the first thing to do was to settle on your list of consulting specialists. The old-fashioned requisites of general medicine, namely, courage, resourcefulness, the inner satisfaction of meeting emergencies single-handed, and old-fashioned and priceless common sense are, I fear, being pushed into the background.

It would be highly useful, if it were possible, for each graduating class to be given a course on the present day essentials of the practice of medicine—a working knowledge, as it were. The instructor should have the ability—a rather rare one, I admit—of putting things in perspective, of evaluating the popular medical fads of the day, and exposing the arch traitor of medicine, commercialism. There is more of this, but thinly disguised, than one cares to admit. Almost every new procedure starts with an aura of commercialism. You can pick out for yourself present day examples.

RESEARCH

Every medical faculty suffers from that most typical American disease, a thirst for publicity. Research quickly makes newspaper headlines. But faculty-planned and directed research lacks the Divine fire, often is sterile or delivers a blue baby. Research calls for imagination and the correlating mind. I cannot agree with Wordsworth that "we are all born trailing clouds of glory." Neither can I agree with Toynbee, the historian who at present is making such a stir, when he says that "all of us are born with congenital sin," but I *do believe* that we all are born with abundant curiosity, and curiosity is the twin of imagination. It is the job of the research worker to ask Nature embarrassing questions, like those with which children

continually stump their parents. To repeat, research calls for imagination and the correlating mind. Uncorrelated facts make the old proverb about the hopelessness of hunting in a haystack doubly true. The medical horizon of today is becoming almost obscured by the haystacks of medical facts—I almost said, of temporary medical facts.

The romance and humaneness of medicine flourish but little, if at all, until after graduation from medical school. As one student of mine put it, his graduation from medical school was like "coming up for air."

I have greatly missed the human touch with patients since I gave up general practice, and what surgery came my way in the early days before I switched to otolaryngology. Science is dehumanizing medicine. Specialism can and often is doing the same. The operating hand is cold, and the microscope has a glassy eye.

When I completed this paper, I looked up what I could find in medical literature on the subjects I have just discussed, and was pleased to find considerable agreement with the ideas I have just expressed. The following is what I got from my reading:

STUDENT ADVISORS

Harvard has had student advisors for many years. In response to growing criticism of the methods employed, President Conant, in his annual report just published, stated that a committee has been appointed to review the whole subject. In advising students, much reliance was placed on the various aptitude tests as these were found better that I. Q. ratings. In World War I there were two things which I greatly dreaded; that I should have to take an I. Q. test, and to ride a horse, but I escaped both. I feel that in giving a student advice and guidance, the intuition of the one giving the advice is equally important with the amount of knowledge he possesses.

THE STUDY OF LANGUAGES

During World War II, languages were taught to service men by a short and most intensive method. In spite of the great publicity given to this plan of teaching, I am not aware of its having been adopted in any of our many colleges, but the Army is planning a four years' course in languages.

THE PREMEDICAL REQUIREMENTS IN THE SCIENCES

In my reading, I found that some medical schools feel that they have overdone the amount of science required for entrance to their

various schools. They are beginning to realize that a general cultural background can be as valuable in medicine as an overloaded background of science.

THE SENIOR CLASS OF THE MEDICAL SCHOOL

In the last report of the University of Toronto, the Dean of the Medical School stated that the senior class has what might be called a series of round table conferences with outstanding local practitioners leading them. At these conferences, up-to-date medical subjects are discussed, and the students are encouraged to ask questions on any subject. This is along the line of the suggestion which I made.

THE THREE-YEAR ACCELERATED
GOVERNMENT-SPONSORED MEDICAL COURSE

The Department of Otolaryngology at the Massachusetts Eye and Ear Infirmary is now putting through its second Eight Months' Basic Science Course. One thing about the G.I.'s who have taken the government-sponsored accelerated three-year medical course, and who have not seen any army service, is their immaturity. Their three-year continuous medical course, without a stop to rest or think, has deadened their curiosity and initiative. It was my great good fortune not to have students of this type. Something would have happened to them, or to me, if I had.

I have permission from Dr. Schall and Dr. Mueller to say that they consider the eight months' basic science course too long. Six months, perhaps four, would be sufficient.

I realize that the summary of this paper, namely, that the broader his education, the better the physician, sounds hackneyed and commonplace, but allow me to add that the more basic a truth is, the more often you hear it, and the sooner it becomes a platitude.

In my advocating a liberal education, allow me to add a note of warning, which is, in these days of plastics real leather is getting scarce, and that no one knows what will be done to education by the psychosomatic approach with its new look.

Quite a few years ago, I put at the end of one of my papers:

"If Medicine is not your greatest pleasure—or, to use a commoner word,—if it is not your greatest fun, you are in the wrong job."

The best wish which I can leave with you is that you have as much pleasure in medicine as I long have had.

127 FRONT ST.

LXVII

OTOLOGICAL FINDINGS IN ACOUSTIC NERVE TUMORS

P. E. IRELAND, M.D.

TORONTO, CANADA

The purpose of this discussion is not to present the neurological findings and treatment of the so-called acoustic nerve tumor. It is a study of 92 patients who were operated upon in our University Hospital from 1924 to date. These cases were all proven, by pathological examination, to be acoustic neuroma. This survey was done in conjunction with the Neurosurgical Service of the Toronto General Hospital in order to ascertain what useful information could be obtained from the careful observations and findings of the otologist.

Although this type of tumor constitutes only between 8 and 10% of the total number of brain tumors operated upon by this Service, it is felt that perhaps this lesion offers the best prognosis, from an operative standpoint, of any intracranial tumor. The complete removal is now relatively assured and because no cortical destruction is entailed, the patient has a postoperative course and recovery free from the troublesome fits or convulsions so often found in meningioma or the other types of favorable tumors. The exact localization of this subtentorial lesion is important to the neurosurgeon in order that a direct approach, in the sitting position, is possible, whereas in other posterior fossa lesions he prefers the face down, suboccipital exposure. The early diagnosis before the impairment of vision or extensive involvement of the various cranial nerves is important. An attempt was made in this analysis to find in what way we, as otologists, might have been of more aid in the problem of localization.

Pathology. The pathology of these tumors has always been a subject of discussion and opinions will vary with the textbook which is consulted. I have no intention of entering into this controversy but will leave it to the expert opinion of the neuropathologist.

I would like to quote from an excellent article in the British Journal of Surgery by H. Alan Skinner:[2]
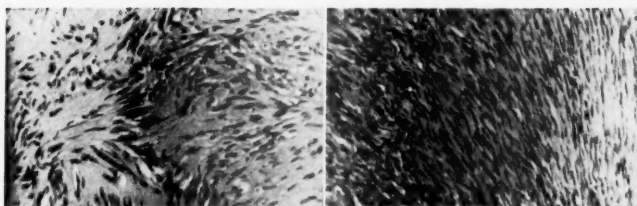
Fig. 1.—Photomicrograph of acoustic nerve tumor showing the palisading effect of arrangement of the fibroblast nuclei.

Fig. 2.—Photomicrograph of acoustic nerve tumor showing the fibroblasts arranged in thick band-like structures.

1. Acoustic nerve tumors arise on the vestibular divisions of the eighth cranial nerve, usually distal to the plane of the porus acusticus internus.

2. The type cell of the tumor is the neurilemma sheath cell.

3. The central, glial segment of the nerve is never primarily involved in the tumor.

4. The somewhat exact position of their origin in the peripheral segment is dependent on peculiar factors in the embryological development of this nerve.

5. The fibrous tissue, generally present in large amounts, is in the nature of a tissue reaction.

6. The tumors may be divided into two types, a cellular type and a fibrous type.

7. The type is determined by the ability or inability of the fibroblasts to confine the tumor cells.

I do think there is some variance of opinion with regard to accepting this in its entirety. It is, however, a working pathological picture which is useful for clinical purposes.

Symptomatology. Cushing[1] in his splendid monograph lays great stress on the "chronology" or order in which the symptoms of an acoustic nerve tumor appear. Deafness of the perceptive type and tinnitus are mentioned as the earliest symptoms observed. There may follow a long latent period before the development of the more localizing signs. We should, however, be prepared to recognize these signs when they do appear. I quote from Cushing: "Certainly

during this [latent] period we can at least spare the patient meddle-some surgery in any effort to improve the hearing."

True peripheral type of vestibular disturbances are rare be-cause of the gradual development of vestibular hypofunction. Cerebellar symptoms are usually the next to appear. These consist of a cerebellar type of nystagmus with mild inco-ordination. The inco-ordination is manifested by an unsteadiness or insecurity on turning or walking, rather than a true objective or subjective turn-ing type of vertigo. The more helpful localizing symptoms of the involvement of adjacent nerves may accompany this cerebellar dys-function or appear at a later period. The sensory division of the fifth nerve is the most common and early nerve to be affected. This is shown by a diminished corneal reflex on the same side. The seventh nerve, although more readily exposed to the pressure of the tumor mass, seems to be less often involved. This may be because motor fibers are less vulnerable to a pressure which is gradually in-creased over a long period. The sixth, ninth, tenth and twelfth nerves can all be involved in a large tumor.

The classical picture is that of a patient with a long period of unilateral nerve deafness, with cerebellar signs and a diminished corneal reflex. Complete loss of hearing function and caloric re-sponse are likely to be present at this time as well as evidence of choking of the discs. Unfortunately, all cases do not present this classical picture. I would like to quote Dr. Cushing again: "But our [neurosurgeons'] mistakes, as will be seen, are more often from in-correct localization, whereas yours [i.e., of otologists] are more often due to the failure to recognize the presence of a tumor at all."

In our series of cases, deafness was the most outstanding single symptom, yet in 14 out of 92 proven cases there was no clinical history of this complaint. Tinnitus was found in only about one-half of those patients with the complaint of deafness (40 in 77 cases). This symptom might precede the deafness but usually was not of as long duration as can be seen by later figures. The tinnitus, differing from that in Ménière's disease when the eighth nerve is resected, did not persist after operation.

Vertigo was a rather late symptom in this series and only in two cases were there the whirling, explosive attacks of the rotation type usually associated with the diagnosis of hydrops of the labyrinth. In both these cases other findings made this diagnosis unlikely. One patient had complete nerve deafness and complete loss of caloric response. The other had complete nerve deafness but incomplete or hypotonic caloric response. This latter patient had diminished cor-

neal reflex and other positive findings. It would seem that there should be no real confusion between Ménière's symptom complex and this lesion. Labyrinthine disturbance, during the slowly progressive loss of vestibular function, is usually not clinically of enough importance to be noticeable to the patient. Facial paralysis occurred in a moderate number of cases but was usually of the mild type of paresis only. Additional information was obtained in many cases by air studies of the position of the fourth ventricle and various views of the internal auditory meatus. I do not think these come within the scope of this paper.

The unfortunate discovery of everyone who attempts to review a series of cases is that a good many of the histories do not contain the information one would wish to find. Some of this was found to be due to a hurried examination and also to the inadequate staff one had available during a six-year war period. In many of the early cases it was felt that we did not know what was of importance, and also at this time adequate hearing tests were not available in order to make a proper assessment. In the 92 cases reviewed we had only 78 in which the patients actually complained of deafness. Thirteen gave no history of deafness and one complained of this symptom in the other ear. This does not mean that 14 patients were not deaf for they might have neglected to notice it or believed it to be unimportant. Most of them were asked specifically in the functional inquiry and answered in the negative. This group will be discussed later in more detail. Of the group of 77 patients who complained of deafness the average duration was for 3.3 years. The extremes of this were that 10 had deafness from ten to eighteen years and 14 had deafness for less than one year. Two were confusing, in that they had chronic suppurative otitis media on the same side.

Before proceeding to a closer analysis of the 78 cases of a proven tumor with deafness as a symptom, we have had to restrict our report to those patients which were considered to have had an adequate ear examination with proper hearing tests and vestibular examination. This does not mean that the neurosurgical department was ignoring the otologist but in most of these cases the diagnosis was obvious and only rough clinical tests of hearing were recorded. Unfortunately this does not help in a statistical study. We are limited in this group to 33 cases.

CASES WITH DEAFNESS AS A SYMPTOM AND ADEQUATE OTOLOGICAL EXAMINATION—33 CASES

AVERAGE AGE: 42 years
    Extremes: 20-34 years (3 cases)
              60 and over (2 cases)

COMPLAINT OF DEAFNESS:

Average: 4.2 years (Duration noted in 31 cases.)
Extremes: 4   cases less than 1 year
                     2   cases 15-20 years

COMPLAINT OF TINNITUS: In 17 cases or 50% was it noted.

Average: 2.7 years
Extremes: 2   days
                     7   years

ROTARY VERTIGO: True rotary type vertigo—2 cases.

1. *Caloric Tests.*

> 26 complete loss of caloric response
> 2 partial loss of caloric response
> 5 not tested (discarded below)

i.e., no cases tested had a normal caloric response.

2. *Hearing Tests.*   (Compared with caloric test.)

a. *Complete Nerve Deafness*: 25 cases (3 discarded)

> 20 cases complete nerve deafness.   Complete loss caloric response.

> 2 cases complete nerve deafness.   Marked hypofunction caloric response.

> —
> 22

b. *Partial Nerve Deafness*: 5 cases (1 discarded)

> 4 cases with partial nerve deafness but all with complete loss of caloric response.

c. *Cases without Nerve Deafness*: 3 cases (1 discarded)

> 2 cases with no nerve deafness but with complete loss of caloric response.

d. *Unusual Cases without Nerve Deafness* (3)

> Case 82: Deafness only in opposite ear.
> Case 78: Middle ear deafness—old otitis.
> Case 81: Normal hearing by all tests.

From this group of patients, which we found to be adequately examined, it would seem that the most constant finding is the caloric response.   In no patient tested was there a normal caloric response. All had the complaint of deafness but three did not show nerve deafness of the expected type and one was completely normal to all tests.

Analysis of Group of Patients Who Did Not Complain of Deafness. If we are to believe that there is a chronological development of a fixed chain of symptoms, this group would seem to present the most interesting features in that it contradicts the textbook picture of the disease. Unfortunately, again we must become critical in accepting evidence which is inadequate or not properly recorded. Immediately we must discard six cases which might have had very interesting features. The remaining eight cases only contribute to the already well known fact that medicine is not an exact study. After concluding from the previous survey that the caloric response in that group of 33 cases was the most constant finding in these tumors, we find four instances of a normal caloric response. We also find two patients with apparently normal caloric and hearing tests.

GROUP OF PATIENTS NOT COMPLAINING OF DEAFNESS

14 CASES

1. 4 cases with partial nerve deafness; complete loss caloric response.

2. 2 cases with partial nerve deafness; *normal* caloric response.

3. 1 case normal hearing test with whisper heard 25 feet in both ears; normal tuning fork tests. Normal caloric response. (Audiometer was being repaired and was not used.)

4. 1 case with no nerve deafness, (?) slight low tone deafness, bone conduction normal. Conversation 20+; Weber not lateralized. Audiogram normal. Caloric test normal.

5. 6 cases were discarded because of inadequate testing or clinical test only.

SUMMARY OF UNUSUAL CLINICAL FINDINGS

92 CASES

1. 15.3% of patients did not complain of deafness (14 cases).
2. 5.5% showed no evidence of nerve deafness (5 cases).
     (This included 2 cases from (4) below.)
3. 4.3% showed a normal caloric response (4 cases).
     (This included also 2 cases from (4) below.)
4. 2.0% showed no nerve deafness and normal caloric response (2 cases).

From this it may be seen that over 10% of patients who have a proven acoustic neuroma may not complain of deafness even if specifically asked. Of this group about 2% may be very misleading

Fig. 3.—X-ray film of internal auditory meatus (Towne Position) showing a normal internal auditory meatus and one which is very much enlarged. There is definite evidence of bony erosion in this area. There may be normally some variation in the size of the two meatuses but not the marked difference seen here. Evidence of erosion and calcification is a most essential adjunct in the consideration of an x-ray film as an aid to diagnosis.

in that, even after careful examination, one may find both an apparently normal acoustic and vestibular function. The absence of impairment of one of either acoustic or vestibular function is also a complicating observation. These constitute the difficult diagnostic cases from the otologist's point of view but do not necessarily make the diagnosis an impossibility. Other neurological findings may indicate a subtentorial lesion which is probably localized in the cerebellar-pontine angle region. In order to illustrate these points, four case histories will be given in summary.

CASE 43.—L. B.   Age 45.   Operation 1948.

   Hearing: History of impairment of hearing 5 years previously that lasted six months but improved.

   P.I.:   No complaint of hearing loss or tinnitus.

             3 years staggering and unsteadiness on change of position.

             2 years recurrent nausea.

             4 weeks vertigo and diplopia, no headache.

   Pre-operative Findings:

             Choked discs and nystagmus.

             Marked 5th nerve.

             Marked 7th nerve.

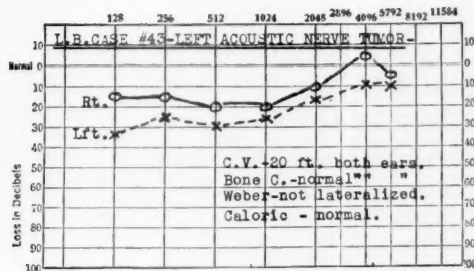   X-ray: Enlarged internal auditory meatus.

Fig. 4.—Audiogram in Case 43.

E.N.T.: Caloric normal both sides.

H.T.: Slight (?) middle ear deafness by audiogram.
Conversation 20 feet+ both ears.
Weber not lateralized. Bone conduction normal.

Comment. Although this case presented essentially negative otological findings it was obviously a subtentorial tumor and the enlarged internal auditory meatus revealed by x-ray examination made this localization likely to be in the cerebellar-pontine angle.

CASE 15.—M. M. Age 36. Operation 1947.

P.I.: No history of deafness or tinnitus.

Unsteadiness and dimness of vision.
No headache.

Pre-operative Findings:

Choked discs.
Nystagmus coarse and irregular.
Unsteady gait, especially on turning.
5th nerve: Corneal reflex down.
7th nerve: Normal.
8th nerve: Normal hearing and caloric response.
X-ray: Enlargement of internal acoustic area.

Comment. Again this patient had obviously a brain tumor in the posterior fossa which could be localized by the fifth nerve involvement and x-ray examination. The otological findings were of no value in the diagnosis.

CASE 78.—E. M. L. Age 33. Operation 1946.

    P.I.:   2 years deafness, tinnitus left ear.

              6 months blurred vision.

              4 months unsteadiness and staggering.

              3 months headache.

Pre-operative Findings:

              Choked discs.

              Unsteadiness.

              Nystagmus.

              5th nerve diminished.

              7th nerve, partial loss.

Otological Findings:

              Old scarred drum left with healed perforation.

              Conversation and whisper normal both ears.

              Bone conduction normal both ears.

              Weber not lateralized.

              Audiogram shows middle ear deafness only left.

              Caloric test: No response after 4 minutes continuous
                  irrigation with ice water.

Comment. The otological report is quoted: "This patient has not the amount or type of deafness expected in an acoustic neuroma. In view of the other signs of nystagmus, unsteadiness with loss of caloric and the accompanying 5th and 7th nerve involvement, I should think the diagnosis of cerebellar-pontine angle tumor is correct with the 8th nerve not involved in the tumor."

The error of not doing a careful otological examination may also be a great disadvantage to the neurosurgeon in the localization of the tumor. One of several cases of error is shown to illustrate this point.

CASE 39.—Mrs. H. A. Age 26. First operation December 1943.
        Second operation January 1944.

    P.I.:   10 months ago pregnant.

              2 months ago nausea and morning vomiting.

              2 months ago frontal headache.

              1 month unsteadiness.

              1 month numbness left face.

              1 month frontal headache.

Pre-operative Findings:

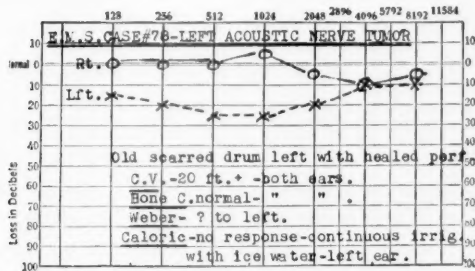   Choked discs.

              Unsteadiness with nystagmus.

128   256   512   1024   2048 2896 4096 5792 8192 11584

E.M.S.CASE#78-LEFT ACOUSTIC NERVE TUMOR

Rt.

Lft.

Old scarred drum left with healed perf.
C.V.-20 ft.+ -both ears.
Bone C.normal- "   " .
Weber- ? to left.
Caloric-no response-continuous irrig.
with ice water-left ear.

Loss in Decibels

Fig. 5.—Audiogram in Case 78.

> 5th nerve markedly diminished.
> 7th nerve marked paresis.

Deafness: No deafness by clinical test.   Caloric test not done.

First Operation: Lying down, occipital approach: Could not expose cerebellar-pontine angle properly; failed.

Hearing after Operation: Audiogram showed definite unilateral nerve deafness. Patient not well enough for caloric test.

Second Operation: Exposed in sitting position and large acoustic neuroma easily exposed and removed.

Comment. If a careful otological examination had been done before the first operation. the exact localization would probably have been made. This would have made the proper exposure and removal of the tumor possible.

SUMMARY

It is of no advantage to try by statistical figures to disprove accepted medical criteria for the diagnosis of specific diseases. We must, however, realize that patterns are altered by the vagaries of the human body which is not an automatic machine. In endeavoring to aid the neurosurgeon to the best of my ability as an otologist, I have been struck by the fact that the literature and textbooks are full of statements which fail to be confirmed by the proven operative findings, in this particular lesion. Listwan-Susser[3] stated that disturbances in hearing occurred in 100% of cases and facial paralysis in 75% in their series. Our group of proven cases tend to disagree with these figures. The exception does not disprove the rule but rather must we realize that the unusual findings do not preclude an

accurate diagnosis if other clinical signs or findings lead one to a useful assessment of the localization of the lesion.

I do not think we deserve the comment of Cushing regarding the failure of the otologist to even recognize a lesion as a brain tumor. We are usually called in an early stage to see these patients because of deafness. There are many cases of unilateral nerve deafness which are not associated with acoustic nerve tumor. I am not going to enumerate these in this paper. I do think, that in any patient in which this diagnosis is made, one might safely put on his record that this may be the early symptom of an acoustic nerve tumor. The succeeding symptoms of corneal anesthesia, cerebellar signs and dimness of vision would then be anticipated if they did eventually develop.

When one is asked by the neurosurgeon for a consultation in a case which is suspected of being a subtentorial lesion we can only honestly state our findings and opinion. The usual picture of a tumor of the eighth nerve, from the otological point of view, is that of complete nerve deafness and complete loss of caloric response. Due to the pathological picture of a gradual peripheral stretching of the eighth nerve over the tumor mass, there may occur, in a small percentage of cases, unusual findings that may deviate from this general pattern of symptomatology. This may range from a completely normal auditory and vestibular function (rare) to the reaction of either the auditory or vestibular portion of the nerve in a normal response. In this series, findings, other than otological, made the proper exploration of this area essential in spite of the seemingly confusing picture.

The neurosurgeon now realizes that in most cases the otologist can be of great value in the localization of these tumors. It is a great advantage to him in the doubtful cases to have an accurate estimation of vestibular and auditory functions. This can not be attained by the rough clinical tests he has at his disposal but must be done by the most careful hearing tests, with the audiometer included, and the proper evaluation of vestibular responses. It aids him in an accurate diagnosis and localization which is important for the proper approach and complete removal of these tumors that constitute one of the most satisfactory operations in his specialty.

CONCLUSIONS

1.   A review of 92 cases of proven tumor of the eighth nerve has been presented.

2.   The otologist must assume the responsibility of the recognition of additional neurological symptoms in cases of unilateral nerve deafness which may later prove to be an acoustic neurinoma.

3.   The examination of the auditory and vestibular portions of the eighth nerve may aid greatly in the localization of subtentorial brain tumors.

4.   The otological findings do not follow the prescribed pattern in a certain number of proven tumors of the eighth nerve.

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DISCUSSION

DR. BENJAMIN H. SHUSTER (Philadelphia):   If Dr. Ireland has called our attention to one fact only, that when we have a patient with deafness we must think of acoustic tumor as a possibility and examine him for that purpose, he has done a great service.

There are two other points I would like to take up in this discussion. The first is the question of normal hearing and normal vestibular responses in the case of an acoustic tumor. I have yet to encounter my first case. I can see that it is possible, but is not likely to occur frequently.

The other point is the question of vertigo. I have always considered vertigo of the rotational type something striking either the vestibular nuclei or internal ear proper suddenly.   Brain tumors and arteriosclerotic changes come on slowly and do not give us the rotational type.

A third point I wish to discuss is that I was rather disappointed that Dr. Ireland did not give us any of the details of the neurological examination and of the caloric tests, which lead to the detection of acoustic tumor.

Most of the so-called newer methods of examination, such as the alternate hot and cold caloric test, give us little information. A mass caloric test with cold water gives a lot of information if you take into account not only the reaction of the affected side but the planes of the canals and the fibers that go off from the canals to the brain.   The vertical canals must be tested especially on both sides as well as the horizontals.   A certain amount of influence is exerted upon the opposite side by any lesion in the brain.   The report today merely refers to the reaction of the side in which the lesion was present.

At the University of Pennsylvania, where I do most of the neuro-otological work, the neurologists and neurosurgeons at first looked disparagingly on the neuro-otological examination.   Now there is not a case of acoustic tumor or cerebellopontine angle tumor in which they would not delay operation until

the neuro-otological examination had justified the diagnosis. I want to mention some cases to indicate why they changed their attitude.

One patient came in with facial paralysis and some impairment of hearing. This patient was sent to the neurologist on account of the facial paralysis. This patient showed a dead labyrinth, and on examining the opposite side repeatedly by caloric test there was no response from the vertical canal. I told Dr. Yaskin that although this patient was not very ill, there were definite signs of an intracranial acoustic tumor indicated by the vestibular tests. He said, "It may be so and I am going to take a chance and operate." He found a tumor the size of a pea in the internal auditory meatus, confirmed later on pathological examination.

Another case that I had was of developing nerve deafness. There were definite vestibular signs of an angle lesion. There were not sufficient neurological signs to make the diagnosis. The operation had been postponed from month to month and year to year, until the patient became so incapacitated that operation was decided on. Von Recklinghausen's disease involving both eighth nerves was found. This case went on for six or seven years after I had told them that there was an angle lesion on one side.

Another interesting case was one in which the opinion was held that there was an acoustic tumor, although the vestibular test showed a fairly good response on caloric examination as well as some hearing. I stated that I did not believe there was an acoustic tumor. It proved to be a case of primary cholesteatoma of the mastoid.

It is simple to make the diagnosis of acoustic tumor if you take into account all portions of the vestibular apparatus. It is such a definite entity, a dead labyrinth on the affected side, with some vestibular involvement on the opposite side. A dead labyrinth alone could hardly be sufficient to make a diagnosis of acoustic tumor. It has to have some intracranial aspects, some interference with the nuclei or their fibers coming off the opposite side.

For these reasons I thought Dr. Ireland should have emphasized the complete examination by means of the caloric test.

Dr. H. I. LILLIE (Rochester, Minn.): I wish to talk to the point of the complete vestibular examination as suggested by Dr. Shuster.

I was interested in neuro-otology early and took the course with Jones and Fisher at the University of Pennsylvania, I think in 1921. We have had the same experience with converting the neurologists and neurosurgeons to the importance of the neuro-otologic examination in conjunction with suspected cerebellopontine angle tumor.

Interestingly enough, the same experience that has been had by us has been had by Dr. Ireland. I think it was in St. Louis in 1923 that it was said that the typical picture of neurofibroma or cerebellopontine angle tumor was the complete loss of hearing, complete loss of function of the labyrinth on the homolateral side and loss of function from the vertical canal on the opposite side. In the group of patients that we examined that was generally true but there were a number of proven cases in which the loss of hearing was not complete and in which complete loss of the labyrinthine function was not present.

I would like to ask Dr. Ireland if he has the percentage with loss of the corneal reflex. That is perhaps the first sign that the neurologists expect to see in a tumor of the cerebellopontine angle; also the incidence of the loss of function in the vertical canal on the opposite side would be of importance.

Dr. JOHN J. SHEA (Memphis, Tenn.): Acoustic neuromas constitute about 8% of all verified tumors of the brain, but a bilateral acoustic neuroma is rare. To add to the splendid presentation of Dr. Ireland, I wish to report such a case.

Miss O. M., aged 20, with a negative family history, was examined as a part of a general neurological study in March, 1920. Her past history had been uneventful except for an acute poliomyelitis in 1915.

Physical Examination: General appearance was that of a well developed and nourished young lady; she was mentally alert and answered all questions with precision. Head was of normal contour and no scars were present. Eyes (Dr. E. C. Ellett): Pupils were dilated, but equal and reacted to light and accommodation. Vision O. D. 15/30; O. S. 15/25. There was a choked disk of 2 mm with normal fields. The nasal membrane was congested and there was postnasal discharge present. Tonsils were buried but clean. Ears were normal. The functional examination showed complete nerve deafness, as she could not hear any of the forks by either air or bone conduction. The Galton whistle could not be heard and the Barany noise apparatus failed to register. Heart, lungs and abdominal organs were normal. Extremities: There was a right ankle and wrist drop. Neurologic (Dr. B. F. Turner): There was flaccid paralysis with atrophy of the extensors and external rotators of the right forearm, and weakness with atrophy of the peroneal muscles of the right leg. Sensory: There were no areas of anesthesia or paresthesia. Reflexes were normal except for the wrist and ankle drop.

Barany tests: Summary: There were no spontaneous findings. Rotatory tests: She appreciated that she was turning, but had no after-nystagmus or vertigo. Caloric tests failed to produce any reaction. Laboratory test: Urine and blood were normal. Both blood and urine gave a negative Wassermann reaction.

Dr. R. Eustace Semmes did a left subtemporal decompression on March 27th. The dura was tense and the brain bulged. The descending horn of the left lateral ventricle was tapped and fluid escaped under great pressure. A lumbar puncture was necessary to reduce the pressure sufficiently to close the dura.

The patient was discharged from the hospital on April 8th after an uneventful operative recovery with slight improvement of vision, but no improvement of hearing.

A second Barany examination on October 8th showed no change in the reactions. On October 18th she was re-admitted to the hospital with vision reduced to light and moving objects. The patient was given an intensive antisyphilitic treatment. Dr. Semmes injected the ventricles; the radiogram showed the skull to be very thick, and there was an internal hydrocephalus. The internal auditory canal was dilated on the left side. This decided the question that the tumor was subtentorial. Dr. Semmes on October 20th did a bilateral suboccipital exploration, and uncovered a tumor in the left cerebellopontine angle. The operation had to be abandoned because the patient took a turn for the worse, but it was hoped that the tumor could be removed at a second operation.

On October 22nd, her vision improved and she could recognize individuals, and on the morning of the 25th she apparently was progressing favorably, but during the afternoon developed a pulmonary edema and died.

Autopsy Findings: There was a healed scar in the left temporal region and a healed "cross bow" incision in the suboccipital region. The skull was very thick and dense, and the porus internus enlarged on the left. The dura was studded on its inner surface with small, soft, opaque white tumors, varying in size from 0.25-1.0 cm in diameter. Eight of these caused marked indentations of the cerebral cortex.

There was a hard, encapsulated tumor in each cerebellopontile angle, measuring 2.5 cm in diameter on the right, and 3.5 cm diameter on the left. The surface was smooth, and the tumor on the left side presented a bossy contour. On the left side there was an elongated bulbous enlargement of the eighth nerve, measuring about 0.5 x 0.25 cm.

There was marked compression of the pons and medulla, greater on the left, distortion of the lobes of the cerebrum and cerebellum, and marked herniation of the cerebellum into the foramen magnum.

The third and lateral ventricles were enlarged and the lumen of the infundibulum dilated.

Microscopic examination showed the typical picture characteristic of dural fibromata (neurofibroma). Many of the smaller growths near the superior longitudinal sinus showed calcareous deposits.

Permission for general autopsy was not obtained.

Summary of Findings: Skull very thick. Multiple neurofibromata identifying cerebral cortex. Bilateral cerebellopontine angle tumor and bulbous enlargement of the left eighth cranial nerve. Internal hydrocephalus (secondary). Compression of pons and medulla with distortion of the lobes of the cerebrum and cerebellum, and herniation of cerebellum into the foramen magnum.

DR. HAROLD G. TOBEY (Boston): Dr. Shuster and Dr. Lillie anticipated me somewhat in emphasizing the importance of the examination of the opposite side and the lack of response from the vertical canal. I think Fisher and Jones offered an explanation for that years ago.

As a word of caution, we have found that the neurosurgeons are extremely loath to operate on the otological findings above. They want other signs, such as the loss of corneal reflex, headache and the vertigo. I think that Dr. Ireland's report today showing that these may be present without any otological finding is extremely important. The question is: Can we prevail upon the neurosurgeons to operate on our findings alone?

I have two patients who have shown typical caloric and hearing findings for ten to fifteen years without any further progress since the disease. The question is whether they have a small tumor which has not progressed. One patient for ten years and the other for fifteen years have shown the typical deafness, tinnitus, lack of caloric responses on one side and from the vertical on the other without further progress.

DR. WILLIAM J. McNALLY (Montreal, Canada): Dr. Ireland very kindly sent me a copy of his paper, so I had the privilege of studying it before coming here.

It seems to me that this interesting paper really opens two aspects of the diagnostic problem. One is our responsibility to the patient who comes to us, who has never seen a neurosurgeon who might warn us of the fact that there is a problem of brain tumor or eighth nerve tumor.

We should always be prepared to at least suspect the presence of the eighth nerve tumor. That demands from us not brilliance but certainly thoroughness in our examination. Dr. Ireland has pointed out that one of the first signs is deafness, so that the patient will come to us usually with deafness. He may not complain of vertigo at the outset but if we have unilateral deafness we should do a caloric test. If we find something that arouses suspicion we should then insist upon a complete neurological examination. In other words, I feel the paper makes a plea for thoroughness in our examination. In addition to the hearing test we should always include caloric tests.

The other thing I would like to say is this: I agree with Dr. Shuster and with these men who emphasize the importance of a complete vestibular examination, but even if we don't do a complete otological or vestibular examination, even if we don't understand what a complete vestibular examination means— and there are a lot of us who don't—if we do a vestibular examination using cold water, it will give much information that may help us and the neurosurgeon.

DR. EDMUND P. FOWLER, JR. (New York): I have two or three questions to ask Dr. Ireland.

First, he made a statement in the early part of his remarks that the surgeons made complete removal of the tumor. He then went on to describe the tumor growth from the region of Scarpa's ganglion. I would like to know if the surgeons in Toronto remove the tumor from the internal auditory meatus.

The second question I would ask is, was it true in this series that the spinal fluid protein was one of the more important signs of cerebral and pontine angle tumor? Our neurosurgeons feel that a high protein is a tremendous help.

Lastly I would like to call attention to a recent article in the British Medical Journal by Dr. Hallpike, who has reported that there is no recruitment in cases of cerebellopontine angle tumor in which there is hearing. As Dr. Ireland pointed out, there are very few cases with hearing, but if all of us will get together and do the loudness-balance tests in the cases with the hearing, we will be able to substantiate or disprove the rather interesting idea that if a nerve lesion is peripheral there is no recruitment, whereas if the lesion is in the end organ there is recruitment.

Dr. PERCY E. IRELAND (Toronto, Canada): I would like to point out that although a good many of these cases were worked up thoroughly, with suitable examination of vestibular responses, they were done by a variety of men, and findings, therefore, were not always consistent. The most you could interpret from them was whether the vestibular response was normal, hypertonic or absent. You have to remember that some of these cases were done 25 years ago.

In answer to Dr. Fowler, we are trying in all cases with hearing to see if recruitment is present. One of these cases did not show any evidence of recruitment.

Our neurosurgeon does attempt a complete removal and therefore sacrifices the facial nerve readily and does a facial anastomosis afterwards.

I have not spoken of a good many of the signs, one of which is the total protein in the spinal fluid. One of our neurologists feels that is a very important sign.

HAZARDS OF INTENSE SOUND AND ULTRASOUND

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Ultrasonic vibration, usually shortened to "ultrasonics," refers to sound waves at frequencies above the upper limit of human hearing. The American Standards Association sets 15,000 cycles per second as the arbitrary dividing line. Sound waves at ultrasonic frequencies differ particularly from audible sound waves in their shorter wave length, in forming relatively sharp sound shadows, and in being attenuated more rapidly as they travel through air.

Ultrasonics in nature are relatively weak. Familiar man-made sources are the jingling of small pieces of metal, such as a bunch of keys, and, of more practical importance, the escape of compressed air, the aerodynamic noise of aircraft and the exhausts of jet engines and rockets. It is with the development of the latter to their present tremendous power that ultrasonics have become a matter of practical concern to the otologist and the flight surgeon. Are these high-frequency high-intensity vibrations a threat to the ear, or to any other organ of the body? And if the ultrasonics are harmless, what about audible sound at the very high intensity levels of 150 db or higher that have been measured near certain modern power plants for airplanes?

Fortunately, as far as general effects from ultrasonics in air are concerned, man has an important protection in the reflecting power of his skin. At 6000 cycles per second, for example, human skin absorbs only about 0.2% of the acoustic energy that falls on it. Absorption is even less at higher frequencies. These values have been determined experimentally for small areas of skin[1] and certainly give the correct order of magnitude. Furthermore, they probably repre-

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sent the maximum absorption for the human body because in any ordinary sound field some of the body surface will be partly protected by the sound shadows to which we have already referred.

In contrast to the good reflection of the human skin, furred animals absorb 12% of high frequency sound energy (a little more or less according to frequency). The energy is turned into heat and the heating may be sufficient to kill a rat, guinea pig or mouse, in a few minutes at intensities of 160 db or thereabouts. Man's clothing lies somewhere between bare skin and an animal's fur. Its absorption for acoustic energy depends on the material and the weave. In general, however, although we can cook a small animal with the energy of a sound field, it is probably impossible to cook a man. Not only does he absorb less energy but he warms up more slowly on account of his larger ratio of mass to surface and finally he has, in his ability to perspire, a more effective temperature-regulating mechanism than the rat or mouse.

We may glance for a moment at the therapeutic possibilities of very high frequencies of the order of a million cycles per second. Such ultrasonics in order to be applied efficiently to the human body are generated in a fluid, usually oil, and transmitted through fluid or solid to the tissue we desire to treat. Beneficial effects for certain forms of cancer and for neuritis have been claimed by German investigators. The design of an "ultrasonic applicator" is given in an article by Theissen of the Acoustical Laboratory of the National Research Council in Ottawa.[2] It is premature to express a final opinion on the therapeutic claims involved. Certainly, however, it is too early for general exploitation of the method. The devices will presumably be evaluated by the Council on Physical Medicine and it is good to know that this problem is being studied by several responsible groups including the Mayo Clinic. We know definitely that sufficiently intense ultrasonics in this frequency range can cause changes in tissue and probably by mechanisms other than mere local heating. It is still uncertain, however, how much margin of safety there may be between the beneficial effects on diseased or cancerous tissue and injurious effects on surrounding normal tissue. It seems likely that the margin will be small and that the practical usefulness of ultrasonics in therapy will be sharply limited in consequence. We may watch these developments with great interest, but at present the burden of proof is still on those who claim that ultrasonic energy can produce therapeutic effects that are not equally well achieved by other available methods.

So much for the general effects of ultrasonics. There is no evidence that air-borne ultrasonics can produce any specific direct

effect on the brain or other parts of the nervous system.[3]   In spite of many suggestions of such mysterious possibilities, the only reliably established effects are either through the stimulation of the ear or some other sense organ.   In general, the same statement applies to the audible frequency range.   At very low frequencies, and as we merge into the problem of mechanical vibration, direct mechanical effects of physiological and pathological importance begin to appear.   Mechanical effects of this sort, whether they be the rupture of an eardrum or the production of neuritis by the handle of a violently vibrating pneumatic hammer, are usually quite obvious.

As soon as we begin to discuss the hazards of sound and ultrasound, we find that the intensity scale becomes far more important than the frequency scale.   It is not the high frequency but it is the high intensity that does the damage.

By "intense" sound in this context we mean, roughly, sound that is strong enough to be perceived by some sense other than hearing. For air-borne sound over most of the audible range the practical threshold of nonauditory perception is somewhere about 120 db. At this level air-borne sound stimulates significantly the sense of touch, especially if it is assisted by *resonance* of some particular structure.   The partly opened mouth or the nasal cavities or sinuses may resonate strongly and greatly surprise the unwary subject in a strong sound field.   Abdominal tactile sensations, perhaps mediated by the pacinian corpuscles, may begin at considerably lower intensities, especially for low frequencies, but this threshold has not yet been properly measured.   Nevertheless we may take 120 db as a significant dividing line.   Here we really begin to *feel* sound and also the ear begins to experience discomfort.   Also at about this level temporary hearing loss is produced by exposures of the unprotected ear for more than a very short time.

Another important intensity level is 140 db.   This is our best present value for the average threshold of pain in the unprotected ear.[4]   Also the sensations from other sense modalities, particularly touch, begin to get uncomfortable.   But as yet, except for local heating effects, we have not experienced pain other than in the ear from intense sound fields, although in the course of our experimental work we have personally been exposed to intensities up to 160 db over a wide range of frequencies.

We have no new information to report on the very important problem of possible cumulative effects on hearing of repeated exposure to intense sounds.   Certainly, a very considerable temporary deafness may be incurred repeatedly without demonstrable cumulative effect.[5]   On the other hand, a partial hearing loss greater than would

be expected on the basis of age alone seems to be statistically evident among workers in very noisy industries including certain branches of military service. It is possible that many cases of definite hearing loss may be due to single exposure to a louder-than-usual sound and not strictly to a cumulative effect of more moderate exposures. Here is a problem on which much more work is needed and which is particularly appropriate for study in the military establishment.

We have two interesting but isolated observations to report that we have made in the sound room at the Aero-Medical Laboratory. One of the authors has an abrupt high-frequency hearing loss. The highest pitch that he can hear corresponds to 6000 c.p.s. In a sound field of 153 db on the average (with peaks at 156 db) at 10,000 c.p.s. he heard a tone, but the pitch was still that corresponding to 6000 c.p.s. The tone was clearly audible, but not in the least uncomfortable although he felt a mechanical turbulence (probably air currents) and some warmth in the ear canal. For the normal ears of other observers this tone was acutely painful. This author's thresholds for 6000 c.p.s. and below were not altered by as much as 10 db for any frequency by five minutes exposure to this 10,000 c.p.s. tone. On another trial of 3.5 minutes at 14,500 c.p.s. at 151 db (with brief peaks at 156 db) the result was almost identical. A faint tone of 6000-cycle pitch was heard with no pain or discomfort and no losses of more than 10 db at any frequency.

Another observation is more definitive. It is, we belive, the first case of rupture of a human eardrum under experimental conditions by exposure to a sustained sound of measured intensity. The frequency was 6500 c.p.s., the exposure lasted five minutes, and the intensity varied from 156 to 164 db (average between 158 and 159) depending on the exact position of the subject's head in the sound field. When the subject, another of the authors, who is an experienced observer well accustomed to intense sound, first placed his head in the sound field the pain was so intense that he was forced to withdraw his head for a few moments. He nevertheless kept his head in the field at the limit of the pain he could tolerate. He describes it as "not the ordinary sharp pain from sounds above 145 db, but a new kind that included dull as well as sharp pain." At the end of the exposure there was blood in his external canal and otologic examination showed a small rupture near the center of the ear drum. The hearing in this ear was somewhat depressed above 1000 c.p.s. and was unmeasurable above 8000 c.p.s. (see Fig. 1).

The drum healed quite promptly and well and, as expected, normal hearing for most frequencies returned within a few days except for a small amount of conduction deafness due to the injury
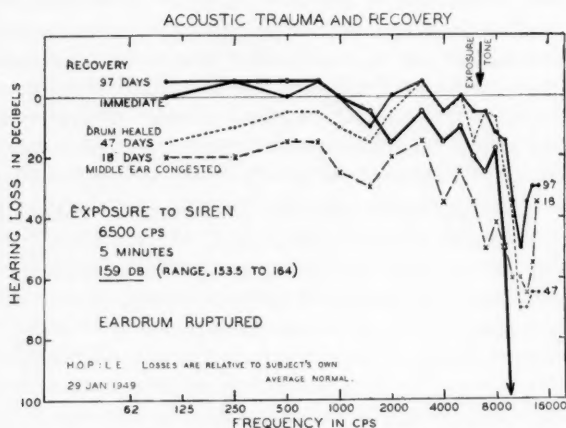
Fig. 1.

and repair in the middle ear. For frequencies above 9000 c.p.s. however, the subject still had an abrupt high-tone hearing loss after 14 weeks. Tones above 11,000 c.p.s. were no longer heard at the upper limit of an exceptionally powerful audiometer. It seems likely that this high-tone hearing loss will prove to be permanent.* We have here the first experimentally determined point on two new contours: (1) The threshold for mechanical injury to the middle ear and also (probably) for the production of permanent hearing loss, and (2) the threshold for "intolerable" pain.

An obvious but good rule is to avoid, except in emergency, exposure to any sound intense enough to cause pain. Less intense sounds for long enough periods of time may cause permanent hearing loss, but painful sounds are certainly dangerous. Even at the threshold of discomfort (120 db) protection for the ears in the forms of earplugs, helmets with ear pads, etc., should be used if exposures are to be of more than a few minutes.

We have pointed out that there is no convincing evidence of any direct effect of air-borne sound or ultrasound on the brain or nervous system. Important effects may be produced however, by

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*An audiogram taken seven months after the original exposure shows complete recovery up to and including 8000 c.p.s. and at 16,000 c.p.s. A dip of 25 db from 10,000 to 14,000 c.p.s. (maximum of 35 db loss at 11,000 c.p.s.) still remains. This represents appreciable further recovery since the latest audiogram shown in Fig. 1.

relatively mild stimulation of a sense organ. A sound does not need to be loud to be extremely *annoying*, and stimulation of the sense of touch may also be very annoying even though it is far below anything that we would be willing to admit is really uncomfortable. Here may be the clue to the fatigue and the irritability and kindred troubles that have been reported repeatedly by individuals exposed to intense sound fields.† In situations reported as causing fatigue and irritability a fairly intense sound is usually present; and most of the sounds, such as from the exhaust of jet engines, contain strong low-frequency sounds as well as ultrasonics. We must remember, however, that in the realm of intense sounds and ultrasonics we are moving rapidly into a new area and we must keep our minds open. Perhaps these very feelings of fatigue, annoyance and irritability are the *first* warnings of a stress of some sort. If we are correct in regarding these subjective feelings as nature's first warning we should not expect to find measurable objective effects or impairment of psychomotor performance until the stress becomes more severe. What we must do is try to standardize and use as endpoints some of these very elusive, but nevertheless real, subjective feelings of fatigue and irritability. They are not hazards in themselves, but they do, in a very real sense, represent a narrowing of our margin of safety. As the first warnings that we may be approaching the limits of our endurance they have a very important meaning. Here is a problem for physiologists, psychologists and otologists alike.

SUMMARY

Present evidence does not indicate that air-borne ultrasonic vibrations constitute a practical hazard to hearing or produce any specific effects on the nervous system or sense organs. In general it is high intensities that are potentially hazardous, not high frequencies as such. Sounds above 120 db stimulate the sense of touch and may cause temporary and possibly permanent hearing loss. Levels above 140 db are painful to the ear and exposures to such levels without special protection should be avoided.

A frequency of 6500 c.p.s. at 159 db caused nearly intolerable pain to one subject and ruptured his ear drum during a five-minute exposure. Some high-tone hearing loss above 10,000 c.p.s. persisted after 14 weeks.

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†Dr. Karl Kryter, in a recent report submitted to the Office of Naval Research, critically reviews our knowledge of the effects of noise on speech communication, hearing and behavior. He points out that *most* persons working in noise (up to 120 db re 0.0002 microbar) apparently became indifferent and "unannoyed" by it; and that nearly all industrial and laboratory experiments which report that noise lowers the work output are open to criticism because of poor experimentation and uncontrolled test conditions.

The vague subjective feelings of fatigue, annoyance, irritation, etc., induced by very intense sound or vibration may represent nature's first warning of the presence of a stress that is not yet severe enough to cause more objective manifestations.

818 SOUTH KINGSHIGHWAY

ENGINEERING DIVISION, HQ. AIR MATERIEL COMMAND.

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LXIX

THE DEVELOPMENT OF THE OTIC CAPSULE IN THE REGION OF SURGICAL FENESTRATION

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INTRODUCTION

Continuing the study of capsular development, reported in last year's meeting of the American Otological Society,[1] the present paper will account for the chief steps in ossification of that portion of the capsule which is the site of surgical fenestration.

The observations are based upon the 30 series of sections selected from more than 300 in the collections at the University of Wisconsin and Northwestern University Medical School. From the 30 series, 8 have been chosen for description and discussion of the process of ossification, especially in the tympanic wall of the lateral semicircular canal.

OBSERVATIONS AND DISCUSSION

In the fetus of 126 mm (16 weeks) the otic capsule is still almost completely cartilaginous. However, at the level of surgical fenestration the first evidence of ossification appears in the region external to the ampulla of the lateral semicircular canal. Here, on the wall of the future tympanic cavity, bone formation begins in ossification center number 3. The first appears in a slightly earlier stage, the last (14th) five weeks later (183 mm).*

*The important steps in the development of the otic capsule, with special reference to the appearance and fusion of the centers of ossification, were earlier described and figured by Dr. Bast.[2] This material, with related information, is contained in Chapter IV of The Temporal Bone and the Ear (Bast and Anson).[3]

150 mm. (18½ wks.)

Sup. semicircular duct

Dura mater

Cartilage

Intrachondral bone

Subarcuate fossa

Cochlear nerve VIII

Vestibule

Utriculus

Crista of amp. l.s.d

Amp. l.s.d.

Lat. semi-circular duct

Cochlea

VIII n.

Perichondral bone

Mesenchyma

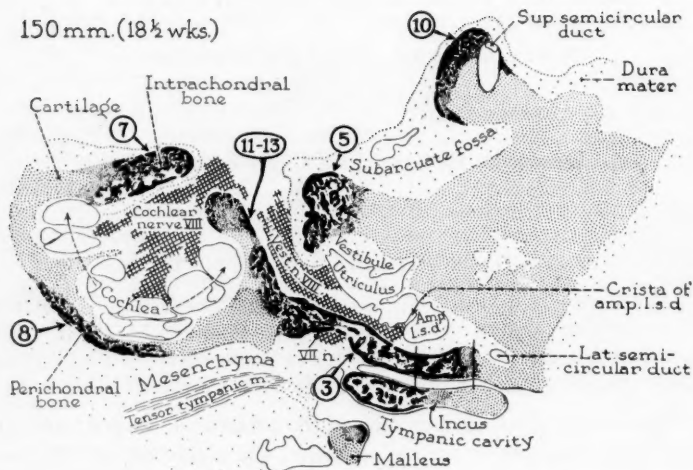Tensor tympanic m.

Incus

Tympanic cavity

Malleus

Fig. 1.—Section of the otic capsule in a 150-mm (18-week) fetus (Wis. ser. 39, sl. 29, sect. 7). X.5. Several centers of ossification have appeared. The three layers of bone are locally present. At this transverse level bone of ossification center 3 has advanced to cover two-thirds of the extent of the tympanic wall of the lateral semicircular canal (see photo-micrograph, Fig. 4a). In this and in the two succeeding figures the area of surgical fenestration is marked by broken lines.

Abbreviations in this and following plates: *Amp. l.s.d.*, ampulla of lateral semicircular duct; *L.s.c.*, lateral semicircular canal; *L.s.d.*, lateral semicircular duct; *M.m.*, mucous membrane; *T.c.*, or *Tymp. cav.*, tympanic cavity; *Tens. tymp. m.*, tensor tympani muscle. Facial and acoustic nerves are indicated by Roman numerals. Layers of bone are marked by Arabic numerals. Encircled Arabic numerals indicate ossification centers (according to Bast).

By way of prediction, it may be said that development of the capsule in the region of the lateral canal, where the surgical fenestra is made, is different from that of the remainder of the capsule in respect to two features: (a) Upon this portion of the capsule the head of the growing incus exerts pressure; (b) here the expanding epitympanic recess and antrum produce an early erosion and thinning of the capsular wall, with subsequent rebuilding.

In the 150-mm (18-week) fetus, periosteal bone is present in the cochlear portion of the capsule, appearing in its apical and basal areas, at centers 7, 8, 11 to 13 (encircled numerals in Fig. 1). In the greater part of the canalicular portion of the capsule, the cartilage remains unaltered. However, on the tympanic wall of the lateral semicircular canal, osteogenesis is, on the contrary, precocious, because of the expansion of center 3 (at encircled numeral 3) lateralward along two-thirds of the canal. Additional centers of ossification have appeared (at encircled numerals 5 and 10 in Fig. 1). On the tympanic wall of the lateral semicircular canal, toward the ampulla (to left) the ossified part of the capsule is now largely excavated and filled with fetal marrow, except for a few islands of the original cartilage (Fig. 4a). These, as constituents of a middle layer, are being converted into intrachondrial bone.

At this point it should be explained that the otic capsule is composed of three layers of bone; these are, from the outside inward, the periosteal (at 1, in Fig. 4a); the endochondral, with contained cartilage remnants (at 2, Fig. 4a); and the endosteal (at 3, Fig. 4a). This inner layer is actually internal periosteal bone, although commonly the term endosteal is employed. The outer layer is formed from the cambium layer of the periosteum around the cartilaginous capsule. It is like the periosteal layer of a typical long bone. The inner layer is always thin and fairly uniform in structure. It is derived from the endosteum, or inner periosteum, and constitutes the immediate wall of the labyrinthine spaces. The middle layer is the most complex in structure; it is composed of islands of altered cartilage (intrachondrial bone) which become surrounded by endochondral bone.

The structure of the middle layer requires brief description. In a stage next in advancement, any high-power microscopic field would be found to contain numerous "spicules." Each spicule is made up of calcified cartilage whose lacunae have been invaded by bone-forming (osteoblastic) cells. These cells deposit a pellicle of bone on the wall of each lacuna; the globules thus formed, with their surrounding cartilaginous matrix, are termed intrachondrial bone.
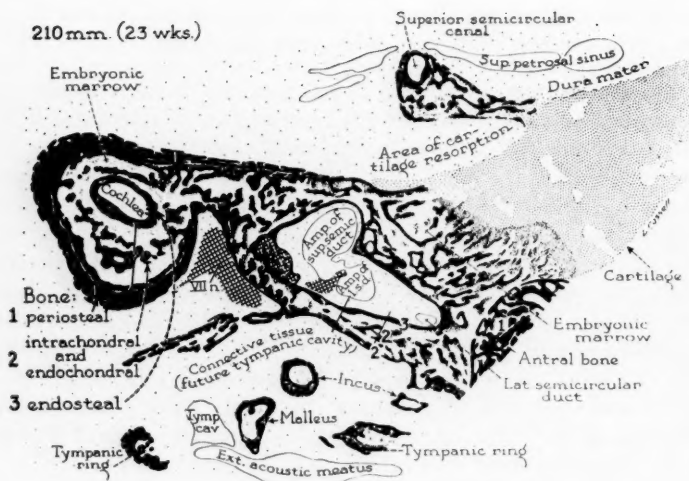
Fig. 2.—Section of the otic capsule in a 210-mm (23-week) fetus (Wis. ser. 51, sl. 56, sect. 4). X.5. In the cochlear portion of the capsule the three layers of bone (numbered 1 to 3, from without inward) form definite and complete zones. In the canalicular part the outer layer (at 1), although incomplete, is a striking feature in the rapidly developing antral area. In the middle layer (at 2) the intrachondrial tissue (cartilage islands) has attained maximum development. The replacement bone, or endochondral tissue, is here minimal in amount. In the intercanalicular region, cartilage is being resorbed in the area of the subarcuate fossa (from the medial side of the capsule), while periosteal bone is being concurrently formed in the area of the developing antrum (on the lateral aspect of the capsule). The cartilage of the tympanic wall of the lateral semicircular canal has been completely replaced by osseous tissue.

Externally, osteoblasts cover these islands of modified cartilage; endo-chondral bone is there deposited. As a result, the islands of cartilage become imbedded in osseous tissue; the intervening marrow spaces are obliterated as the spicules broaden. The intrachondrial bone is not destroyed in the process, but does become less conspicuous as its relative bulk decreases.

Even in a fetus of 193 mm (22 weeks), the external layer (at 1) has begun to undergo erosion, despite the fact that it has been so recently formed (Fig. 4b). Moreover, part of the subjacent middle (endosteal) layer (at arrow) is being removed.

At 210 mm (23 weeks), endosteal bone (at 3, in Fig. 2) has formed a complete shell for the labyrinthic spaces (cochlea, vestibule and semicircular canals); cartilage persists, as a large mass, only in intercanalicular territory, lateral and superior to the subarcuate fossa (Fig. 2). Since no epiphyseal areas are present (as secondary centers of ossification), and since the 14 centers have fused, no fur-ther growth of the capsule will take place; the capsule has attained adult dimensions. Enlargement can hereafter be only peripheral, through the application of additional periosteal bone (at 1, in Fig. 2). The latter stratum is, at this stage, in a condition of active in-crement in the cochlear portion of the capsule. But in the canali-cular portion it is limited to the antral area. Between the inner and outer layers, the middle zone (at 2, in Fig. 2) is one of quiescence. Here the intrachondrial bone is well formed. Endochondral bone is just beginning to appear. Consequently, the middle layer is still loosely textured. In the wall of the lateral canal, erosion has re-sulted in the removal of all of the perichondrial bone, some of the endochondral bone, and the imbedded intrachondrial bone. Thus, while still immature, the wall is being destroyed.

In the capsule of the 270-mm (30-week) fetus, in the coch-lear region, periosteal bone has developed rapidly, to add extracapsular bulk to the fetal temporal bone. Increment deepens the internal acoustic meatus and adds length to the cochlear portion of the cap-sule. The endosteal layer is thin and remains so throughout life. Replacement bone, of the middle layer, is experiencing a surge of development in the canalicular portion of the otic capsule. The "spicules" of this layer are a combination of the two types of osseous tissue, namely, the replacement bone and the altered osseochondral islands, of antecedent formation. Comparable activity is postponed in the cochlear portion until the time when the fetus has reached the stage of term, or during late prenatal life. This is tantamount to saying that during approximately a ten-week period, termination at birth, the production of endochondral bone progresses rapidly in
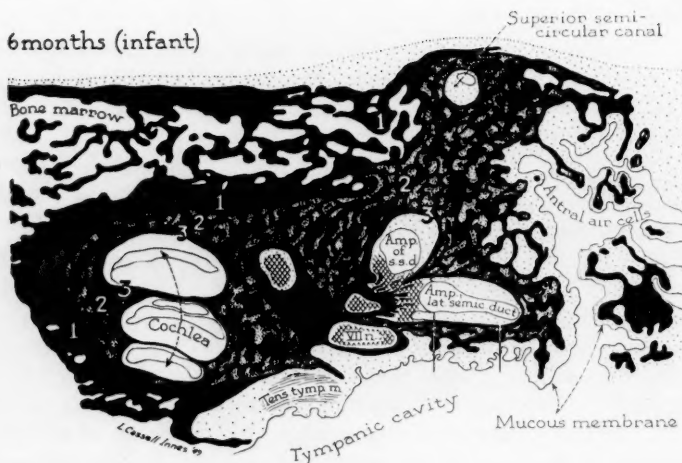
Fig. 3.—Section of the otic capsule in a 6-month infant (Wis. ser. 121, sl. 26, sect. 5). X.5. At this stage the layers of bone have become fused with each other; yet, they remain distinguishable. In the cochlear region, on the medial aspect of the temporal bone, marrow spaces are abundant in the periosteal layer; however, in the deeper stratum of the same layer the periosteal shell of the cochlea (that is, the original capsule) remains petrous in character. Comparably, in the canalicular region, periosteal bone has been removed in favor of antral air-cells. Here the mucous membrane has, in addition, invaded the endochondral bone, pressing into the intercanalicular tissue. The tympanic wall of the lateral semicircular canal is similarly affected, lesser crypts being produced in the canalicular wall. This process has resulted not only in the destruction of the outer (periosteal) layer, but also in the irregular removal of some of the subjacent middle layer (see photomicrograph, Fig. 4c).

the territory of the semicircular canals and vestibule, then to be shifted to the cochlea.   In the canalicular wall there is evidenced a filling in of the middle layer in its deeper portion, with concurrent, continued erosion on the superficial aspect to include the cartilage islands.   The outer layer is wholly wanting.   The inner layer is thin, and always will be.

In a specimen of term fetus, in the cochlear portion of the capsule, the inner layer is fused with the middle stratum; the latter is composed chiefly of endochondral bone, cartilage islands now being inconspicuous.   Extracapsular tissue is thick.   The structure of the capsule immediately around the vestibule is similar to that of the cochlea.   However, the intercanalicular area is still nonpetrous and immature, being made of endochondral bone, with relatively sparse remnants of cartilage islands.   In the region of surgical fenestration, the middle layer is undergoing condensation.   But owing to erosion on the tympanic aspect, the entire wall is thin.

In a 6-month infant, in the cochlear region, in the medial portion of the temporal bone, marrow spaces are abundant in the periosteal layer (Fig. 3).   But in the deeper stratum of the same layer, the periosteal shell of the cochlea (that is, the original capsule) remains petrous in character.   In the canalicular region, periosteal bone is being removed in favor of antral air cells.   On the tympanic surface of the capsule the thin inner layer (at 3, in Fig. 4c) is still indistinguishable from the middle layer (at 2, in Fig. 4c).   The latter is very erose and irregular.   Upon this tympanic surface new bone is being deposited (at 4, in Fig. 4c).   Seemingly it is periosteal in nature, although of secondary formation.   Having been destroyed externally, the lateral canalicular wall is now being rebuilt.

In a 19-year adult, with the exception of the tympanic wall of the lateral semicircular canal, the histological structure of the bone (allowing for individual variation) is essentially similar to that of the fetus at term.   The bone of the lateral canalicular wall is now quite compact (Fig. 4d).   Intrachondrial bone is represented by a few scattered islands.   The tympanic margin of the capsule is smooth. New bone has been deposited to one-half the total thickness of the wall.   Consequently. the thickness of the wall in this region merely equals that of the 150-mm fetus.

CONCLUSIONS

The otic capsule differs from all other skeletal elements of the human body in respect to function, speed of development, schema of ossification, histologic fabric.
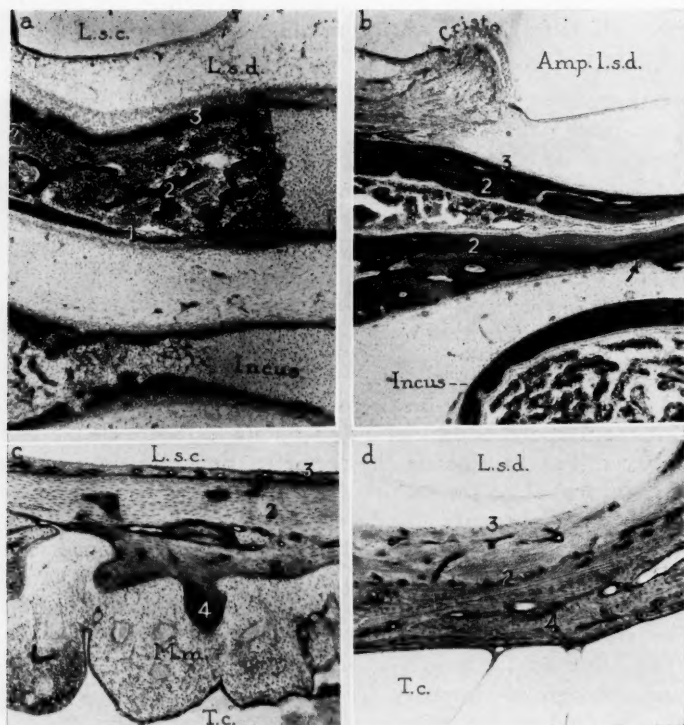
Figs. 4a to 4d.—Photomicrographs of sections of the otic capsule, in the region of the tympanic wall of the lateral semicircular canal.   X.32.

Fig. 4a, fetus of 150 mm or 18 weeks (Wis. ser. 39, sl. 29, sect. 7), in which the three layers of bone are present. Fig. 4b, fetus of 193 mm or 22 weeks (Wis. ser. 85, sl. 30, sect. 7), showing erosion of the outer (periosteal) layer and part of the middle (endosteal) layer (at arrow). Fig. 4c, infant of 6 months (Wis. ser. 121, sl. 26, sect. 5), in which deposition of new bone (at 4) has been initiated. Fig. 4d, adult of 19 years (Wis. ser. 29, sl. 226), in which the newly formed layer (at 4) has attained considerable thickness but not exceeding that of the 150-mm fetus, shown in Fig. 4a.

Functionally, the otic capsule serves as an osseous box to house protectively the organs of hearing and equilibrium.

Its development is dramatically rapid; the capsule attains adult dimensions when the fetus has reached the middle of its intra-uterine existence, whereas a typical long bone continues growth through a period of 20 or more years. The fetal capsule becomes imbedded in thickening periosteal bone.

Constitutionally, the otic capsule is unique. It is made up of numerous (totally, 14) ossification centers. Bone spreads almost concurrently from the core of each of the 14 originally independent centers, not, as in a typical long bone, from a point of initiation in the middle of a diaphysis (shaft) toward terminal epiphyses (at the extremities). Fusion of centers is peripheral; there exist no zones of secondary, or "epiphyseal," growth such as regularly occur in a long bone. Fusion, with complete obliteration of fusion-lines between centers early converts the otic capsule into an osseous unit, a single bone. In this respect it differs from the cranium, whose constituent elements (parietal, frontal, etc.) remain at least partially separable along their boundaries of sutural contiguity.

Histologically, too, the otic capsule is exceptional. Its outer layer, thickened to outbulk the contained capsule, subsequently becomes pneumatized through invasion of petrous bone by mucous membrane. The middle layer, most complex of the three, retains throughout life a considerable fraction of its primordial cartilage in the form of chondral islands imbedded in endochondral bone; once produced, as a tissue of combination nature, it is never remade into haversian bone. The inner layer is simplest; it forms a mere shell for the labyrinthic canals, cochlea and vestibule.

The developmental time-tables of the three layers differ. The three of them are present in the midterm fetus (183-mm) yet they spread at different rates, and in this phase of growth, the cochlear and canalicular portions are not alike. This matter requires elucidation.

The inner, or endosteal layer, is actually complete at midterm (183 mm); it undergoes no increase in thickness and none in size as a covering for the periotic labyrinth. It fuses with the middle layer.

In the middle layer, the intrachondrial bone appears before the endochondral. In both divisions of the capsule, cochlear and canalicular, intrachondrial bone appears in the fetus of 18 weeks (147 mm). It never wholly disappears. This kind of bone attains max-

imum development within a period of 6 weeks (in the 210-mm
fetus). In the cochlear region the intrachondrial islands retain
primordial form for almost a month (to the 246-mm stage). There-
upon, slow formation of endochondral bone is initiated (deposited
upon the intrachondrial islands). At term, the process is suddenly
accelerated, growth continuing through the first few years of in-
fancy with such alacrity that the cochlear region of the infantile
ear closely resembles that of an adult bone. In the canalicular re-
gion there is no such lag in development. From its first appearance,
deposition of endochondral bone is rapid up to the age of 30 weeks
(270 mm), at which stage the adult condition is attained. It is
never replaced by haversian bone. This layer remains distinctive
in the adult.

The outer, or periosteal, layer is complete in the cochlear region
earlier than in the canalicular division of the otic capsule. As an
extracapsular addition, it continues to expand until the time of
puberty, to produce the petrous part of the temporal bone. Be-
ginning in the canalicular region, the periosteal bone of the otic
capsule is invaded from the developing tympanic antrum. In the
intercanalicular area, pneumatic spaces spread beyond the territory
of the periosteal bone into the next layer, namely, the endochondral.
So early is this invasion (beginning in the 28th week, or 246-mm
stage) that the endochondral bone never attains petrous character.

In the surgically important region of the otic capsule, namely,
the tympanic wall of the lateral semicircular canal, the process of
ossification is, in the early stages, the duplicate of that which oper-
ates to form any typical portion of the capsule: the three regular
layers of bone are produced, to replace the primordial cartilage; to-
gether they undergo *no* expansion after once having fused along
their contiguous surfaces. However, at approximately the stage of
midterm, the process of ossification in the region of surgical fenestra-
tion begins to follow a special course. The capsule here consists of
the three typical layers in the fetus of 18 weeks (150 mm). At 22
weeks (193 mm), the recently formed periosteal bone is being re-
moved. So rapid is this process that in a 30-week (270-mm) fetus,
not only has the outer layer been resorbed, but varying amounts of
the middle layer are removed, with some of the cartilage islands.

Following this resorption, new bone is added on the tympanic
surface, to restore a smooth contour. As a consequence, the muco-
periosteal membrane covers this secondary bone, instead of the orig-
inal periosteal layer.
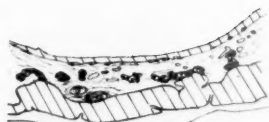
P 4 R ♂ 13 YRS. SL. 260

P 26 R ♂ 68 YRS. SL. 310
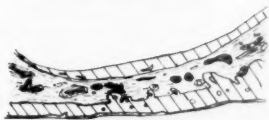
P 25 R ♂ 17 YRS. SL. 295

P 33 R ♂ 50 YRS. SL. 255

P 27 L ♂ 76 YRS. SL. 310

Diagrammatic sketches of horizontal sections through the tympanic bony walls of the lateral semicircular canals of adult otic capsules at various ages. The endosteal or internal periosteal layer of bone is shown at the top of each sketch and is shaded by oblique lines. The other layer of bone is at the lower part of each sketch and is also shaded by oblique lines. In the middle layer the dark mottled islands are intrachondrial bone and the surrounding finely shaded parts are endochondral bone.

And so it is that the tympanic wall of the lateral canal, after having been eroded deeply on the external aspect, is rebuilt to assume regular structure and thickness.

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DISCUSSION

DR. THEODORE BAST (Madison, Wis.): A careful study of large series of adult petrous bones in the region of the tympanic wall of the lateral canal or the region of the surgical fenestra has not been made, but a casual survey indicates that the embryological process of resorption of the middle layer of bone as described by the essayist is much the same at all adult ages. There are individual differences but they are not significant. The accompanying figure shows sketches of the sections through the tympanic wall of the lateral semicircular canal of adult otic capsules at different ages. In all cases part of the middle layer of the original capsule remains. Some islands of intrachondrial bone are preserved in all of them. Many are present in the 13, 50 and 68-year-old and few at the ages of 17 and 76 years. It seems that no changes occur with age. In the 50-year-old, part of the middle layer of bone (to the left) suffered very little resorption and rebuilding, whereas the other part (to the right) was largely removed, even the islands of intrachondrial bone. It is also apparent that the islands are more resistant to resorption and a number of them are seen projecting into the area which was resorbed, but is now filled with secondary bone.

At first it seemed that this resorption and consequent extra activity of the periosteal cambium might be significant as to the relative activity of the endosteal or periosteal cambium in postfenestration regeneration of bone. Since these changes occur during intra-uterine life and no further change occurs postnatally, it appears now that such significance is minimized.

A STUDY IN OBJECTIVE AUDIOMETRY WITH THE USE OF A PSYCHOGALVANOMETRIC RESPONSE

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It has long been recognized that the present methods for hearing evaluation, while reasonably accurate for the great majority of patients tested, have been proved unsatisfactory for determining the hearing in two groups of patients: young children and those with psychogenic deafness. Pediatricians and otologists for years have known that determinations of hearing in children five years of age and under are unreliable. During the past war and since that time, the problem of psychogenic hearing loss has come to the fore, and numerous attempts have been made to develop methods of examination that would give an accurate picture of the actual hearing loss in individuals suffering from what has commonly become known as psychogenic hearing impairment.

At the meeting of the American Otological Society in 1948, several hours were devoted to discussions of modern methods for determining hearing levels, and the conclusion was reached that the principal weakness of our present technique lies in the fact that standard tests depend primarily upon subjective responses. Until some method could be evolved which used objective responses, the audiograms obtained from children and from those patients suffering from psychogenic overlay would be subject to grave questioning. The literature describes many studies along objective lines. Recently, Dix and Hallpike[1] have developed the peep-show technique whereby children can be conditioned to press a button to light a pretty picture in response to a given tone. Michels and Randt,[2] in 1947, showed that auditory stimuli could be recorded with an encephalograph. These, however, were usually at levels well above threshold. In 1947, we became interested in the possibilities of con-

ditioning children to respond to auditory stimuli. After exploring various methods of approach, it was decided to attempt to adopt a skin resistance technique which had been under exploration since 1922 by Dr. Curt Richter of the Phipps Clinic at the Johns Hopkins Medical School. With his apparatus Dr. Richter had been able to demonstrate skin resistance changes following such external stimuli as pin pricks and mild electric shock. We felt that by using an auditory stimulus as a warning signal a few seconds before the electric shock was given a patient might be conditioned to develop skin resistance changes following the auditory stimulus in anticipation of the coming shock. This problem was discussed with Dr. Richter and Dr. John Whitehorn, Professor of Psychiatry at the Johns Hopkins Medical School, who felt from the experience gained with Dr. Richter's patients that there would be no danger of any after-effects in either children or adults who might be conditioned by this method.

Preliminary studies were begun in the winter of 1948. The first patients tested were volunteers whose hearing impairment had been well established with numerous audiograms. The results of this early work showed such promise that a preliminary report was published in the Bulletin of the Johns Hopkins Hospital[3] in May, 1948. To date over 200 patients have been studied by this method and have had skin resistance audiograms made. Except with the infants, standard audiograms have been made immediately after the completion of skin resistance audiometry; thus, we are able to make a close comparison of subjective and objective records.

A description of the equipment used and the technique that has been developed is in order before discussing the results of these studies. It is best to describe the equipment that we use for determining skin resistance changes in the words of Dr. Richter and Dr. Frederick G. Whelan,[4] who have assembled the electronic devices used to magnify changes in skin resistance. Such changes were first described in 1888 by Feré,[5] who discovered that in hysterics a variety of excitations could decrease the resistance that the body offered to the passage of minute direct electrical current (Fig. 1). "It consists of an amplifier (General Radio Company, Type 715A Direct Current Amplifier operated on 110 V. 60 cycle supply), a 5 milliampere ink recording ammeter (Esterline-Angus Model AW), a control panel and a 4½ Volt battery. These parts are mounted on a stand equipped with casters. It is essentially a wheatstone bridge circuit. The voltage supply comes from the 4½ Volt dry battery. The strength of the current that passes through the patient is not constant. It may vary between 2 and 20 micro-amperes, depending upon the resistance of the patient. It is so imperceptibly small that
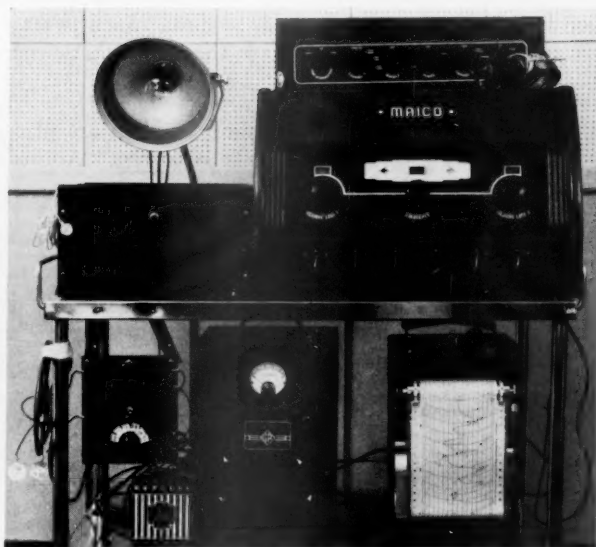
Fig. 1.—Photograph showing all the equipment necessary for skin resistance audiometry. The control panel, direct current amplifier and ink recording ammeter are placed on the lower shelf, while the Harvard inductorium, in its shielding box, can be seen along side of the audiometer on the top of the table. The infra-red light completes the equipment.

it does not stimulate the skin. The changes in the patient's skin resistance as they affect the currents in the bridge can be amplified to various degrees by the amplifier and recorded in ink on the paper recorder." A standard make, well grounded audiometer and a Harvard inductorium coupled with a 6-volt dry cell battery complete our list of equipment.

The skin resistance electrodes leading to the apparatus from the patient are placed opposite each other on the palm and dorsum of the hand or on the sole and instep of the foot, as it has been found that skin resistance changes are greatest on these surfaces. Shock electrodes are placed on the arm, leg or back and the stimulating current is regulated through the Harvard inductorium in circuit with the 6-volt dry cell battery. The intensity of the shock can thus be graduated from each individual and can be changed at any time during the study. As the electrical changes to be recorded vary in proportion to the activity of the sweat glands under the electrodes, one must take care that a patient is warm during the test. When

necessary, we have heated the patients with infra-red lights until perspiration begins to appear on the body surface. Such sweat gland activity is the result of stimulation of the sympathetic nervous system.

Adults are seated in a chair for testing; children are placed on an assistant's lap; both face away from the observers. Adults have the electrodes placed on the hand with the shock electrodes on the arm or leg. Children have the electrodes on the foot with the shock electrodes in the middle of the back or on the thigh. The electrodes are zinc discs which are covered with Kaolin paste in zinc sulfate solution and held in position by adhesive tape. A single earphone is held against the ear to be tested. A selected tone of high intensity is introduced into the subject's ear and this is followed in four or five seconds by an electric shock. The shock is of sufficient intensity to cause the patient some anxiety in anticipating the stimuli to follow. The strength of the shock is varied a great deal with different individuals, as no two people seem to have the same tolerance. Frequently, it is necessary to increase the shock intensity two or three times during an experiment, as the stimulus becomes less effective with repetition. Skin resistance changes in response to stimulation occur about $1\frac{1}{2}$ seconds after the stimulus is given. The method used to condition a subject is to continue introducing the auditory stimulus at a high level followed by the shock, always using the same tone until there is evidence of conditioning (Fig. 2). This is shown as a definite break downward in skin resistance following the tone and preceding

Fig. 2.—Specimen tracing showing the determination of threshold for 1024 d.v. by the skin resistance technique in an individual previously conditioned to auditory stimuli.

the break that follows the shock. At this point one gets two definite breaks on the graphic record. When the subject begins to respond regularly to auditory stimulation, the intensity of the tone is gradually diminished until the threshold is reached. Following each response to the auditory stimulus, the shock is repeated. When the conditioned patient shows no change in skin resistance after auditory stimulation, no shock is given; such a level is considered to be near the threshold. Repeated readings are taken at points above and below the tentative threshold as in routine subjective audiometry. A shock is given following each positive response, but no shock is used when there is no change in skin resistance. Thus, by
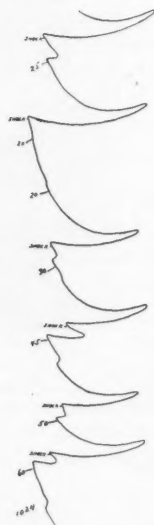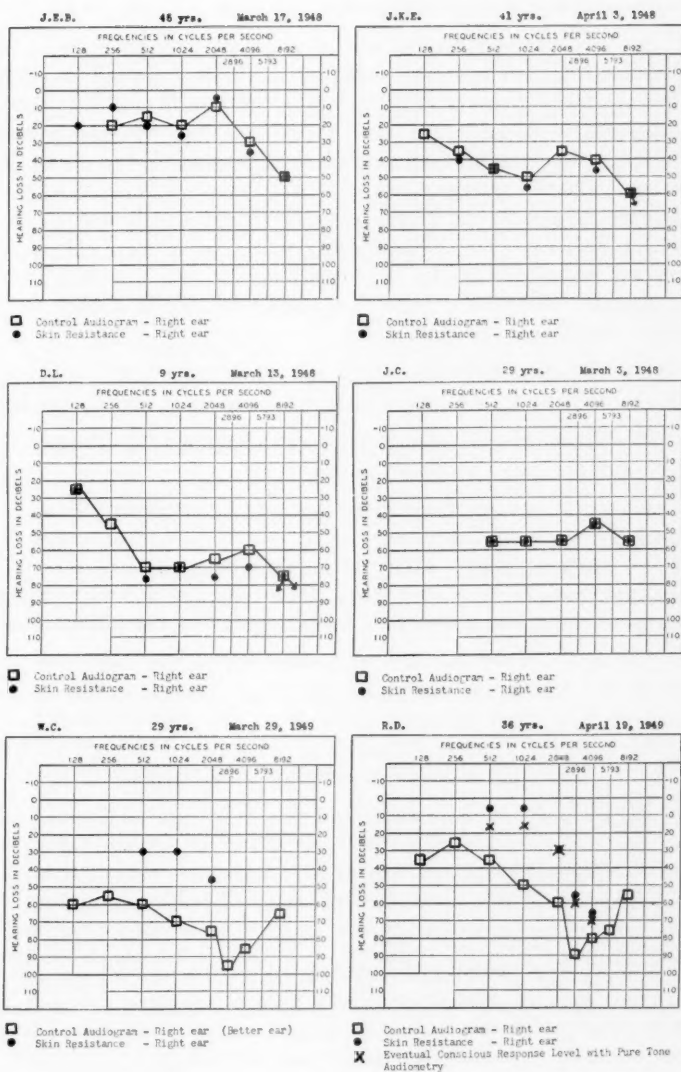
Fig. 3-8.

standard "rising-intensity" technique, the threshold is established for a given tone. The procedure is repeated on other tones until the sound spectrum is satisfactorily explored.

Patients may be conditioned after from two to eight or ten repetitions of high intensity auditory stimulus followed by shock. It has been found that a patient conditioned to one tone is thereafter conditioned to all tones audible to that individual. With individuals suffering from total hearing loss, we have been unable to obtain skin resistance changes previous to the electrical shock. They do not develop the double break in the graphic record which was discussed previously. Persons with profound hearing loss in some octaves will show the normal double break in the remaining audible octaves, but do not show normal conditioned responses where the auditory stimulus is insufficient to register.

To obtain a five-tone threshold audiogram from 500 cycles through 8000 cycles on both ears takes about 45 minutes to an hour.

We have selected the following audiograms to illustrate the types of hearing loss we have studied so far:

Figure 3 represents one of our earliest audiograms. It was obtained from one of the authors in a laboratory with a rather high level of ambient noise. Many previous audiograms had shown the mild high tone loss recorded here. The right ear was used and the controlled subjective audiogram is represented by the joined squares.

Figure 4 shows the skin resistance audiogram compared with the control test on an individual suffering from a hearing impairment of five years' duration. He had a history of well-treated syphilis and was a heavy drinker and a chain smoker. His hearing by bone conduction was much reduced in both ears.

Figure 5 shows the control and skin resistance audiograms on a girl, aged 9, with a three-generation history of severe hearing loss. The child's audiogram and that of her mother are practically identical. They both have moderate reduction of bone conduction. Her past audiograms are essentially the same as those shown.

Figure 6 represents the audiogram of an otosclerotic patient with excellent bone conduction and without tinnitus. She has been under observation for four or five years. This is the most accurate correlation of thresholds we have ever obtained.

Figure 7 presents the studies made on a 29-year-old man who had been in the military service. He had done some artillery firing and also suffered from a fungus infection in his external ears. His subjective audiograms did not agree with the results of his Stenger
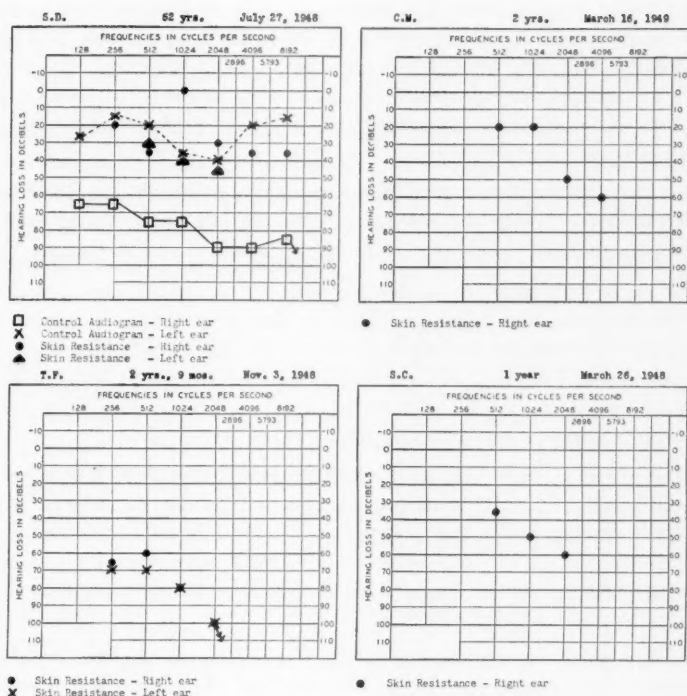
Fig. 9-12.

test, so after repeated subjective tests, the best of which is shown here, skin resistance tests were made on the three tones shown. The discrepancy is obvious.

Figure 8. This 36-year-old man was examined in the Out Patient Department, complaining of severe deafness in the right ear. He claimed he could not hear with his left ear occluded. The control audiogram seemed much better than his speech reponses. Skin resistance audiometry showed a discrepancy, and after this difference was discussed with the patient, another subjective test was done, which corresponded with the objective record.

Figure 9. This 52-year-old man was a seaman. About a year previous to our examination he was struck on the right side of the

head with a loading hook. Following this he claimed severe impairment in the right ear. He was tested in several cities, and the audiograms showed even more loss than our audiograms. Skin resistance testing showed the above interesting crossover of the graph of the left ear. The subjective and objective tests on the left ear were substantially consistant.

The following three audiograms have no controls; they are selected from among the young children we have been studying.

Figure 10 represents a study made on a two-year-old epileptic child with very poor social development whose inattention and lack of vocabulary caused her pediatrician doubt as to the presence of hearing. Her responses, while not representing perfect hearing, established the fact that she should at least have started some speech development and could respond to loud spoken voices, if it were not for a severe mental retardation.

Figure 11. This two-year and nine-month-old child whose hearing impairment had been suspected since the age of one showed very definite responses for the octaves indicated. After our tests were completed the child's mother produced audiograms done on an older brother who is now in a school for the deaf. The two sets of audiograms were essentially identical.

The final audiogram, Figure 12, was one of our earliest attempts to establish the practicability of our method. The child tested was a twelve-month-old youngster recovering from influenzal meningitis. The pediatric department was anxious to establish whether the child's hearing had been destroyed. It was still quite weak when tested in a very noisy environment. We do not feel that this represents threshold audiometry, but it was sufficient proof to reassure the family that the child had not had its hearing wiped out by meningitis.

SUMMARY

A technique is presented here for the achievement of objective audiometry by psychogalvanic skin resistance. The basic measurement of responses through the sympathetic nervous system has been well known for many years, and skin resistance measurement is currently being applied to a wide variety of behavioral events. Our experience with this technique in audiometry indicates that it is probably a valid functional measurement of the peripheral auditory mechanism. As a clinical and a research technique, it offers the benefits of objectivity, requiring no conscious co-operation from the person under test; it therefore gives promise of special use-

fulness in diagnostic work with very young children and adults who will not or cannot respond to subjective techniques.

In conclusion, we wish to emphasize that the technique here described is not free from difficulties. We do not feel that this is the definitive answer to the problem of objective audiometry. We have found serious problems in the control of movements of our children, and we have encountered confusing difficulties from outside electrical interference. We hope, however, that it offers a practical approach to the field of objective threshold audiometry.

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DISCUSSION

DR. MOSES H. LURIE (Boston): In the past three to four years we have been using this type of objective testing for hearing at the Eye and Ear and at the Foundation chiefly with two objectives; one, to determine absolutely whether we have true hysterical deafness or some deaf problems that come to us, and the other, to determine how much young children have in the way of response, especially after influenzal meningitis and similar conditions in which the question of hearing comes up.

As I surmised from Dr. Bordley's paper, he, too, has had difficulty in evaluating some of the results, especially in the young people. With this method, which is done for us by Dr. Knapp who is associated with the Neurological Department at the Massachusetts General, the problem always came up whether the response that we received, especially in young children, could be said to be a response to the auditory centers of the brain or whether it was a reflex action occurring between the cochlea and thalamic region and in that way starting the sympathetic reaction in the skin. That is one of the problems which will have to be solved before we can say that this is a real objective method of determining hearing because in children we cannot get a more or less reflex response to sound without the conscious auditory context being involved in the process. The hysterical adults we have had have all definitely shown a response with the skin test which indicates that the patient was responding to the sound, though he announced that he heard no sound at the time. With proper psychiatric treatment a great many of these cases regained a good deal of their hearing. Whether we will be able to do the same thing with the youngsters is another story.

Right now I am watching a child after influenzal meningitis who gave no response to the skin galvanometer test in the beginning and is now, after six months, beginning to show some response, but still shows no attempt to repeat the sounds to which the mother is exposing her.

DR. HALLOWELL DAVIS (St. Louis): Do the authors feel that there is any problem from wandering of the attention? It will obviously be very difficult to give a warning signal. The question is whether their experience indicates that a warning is at all desirable or necessary.

Another point is, have the authors tried to see whether they can produce false positives, i.e., whether by merely imagining that they hear a sound they can make the machine indicate as though sound actually was heard.

DR. WILLIAM G. HARDY (Baltimore): In answer to the question of a false positive response, we have tried to watch that closely from the onset, but obtained no consistent response. We have also had experience with false negative responses that proved even more revealing. One of our colleagues did his best to induce all manner of plicative interruption in the middle ear cavity, over which he says he has some control. The demonstrated results of the objective test ruled out all the goings-on, for the reason obvious to anybody who has worked with psychogalvanometric deviations, that passing stimuli or passing emotional responses of any appreciable degree naturally show themselves on the instrument and are quite readily interpreted as such.

The problem of wandering attention is tremendous with small children and very possibly is insurmountable.

We initially contemplated the use of associated stimuli on the pleasure rather than on the pain side, using light as a preferred possibility, but the results were completely unstable. I think possibly the best answer we know at present is that the use of an adequate, probably subthreshold, quantity of galvanic shock as the associated stimulus is the best attention-centerer.

In terms of the point that Dr. Lurie raised, I think it would be safe to say that the entire neurophysiologic structure of this method of skin resistance measurement of a wide variety of stimuli has scarcely been touched. Certainly the basic questions have not as yet been answered. I suspect further that what is involved is rather a thorough-going redefinition of the basic concept of the term "hearing." There are many assumptions that are taken for granted until one considers the entire behavioral complex, particularly of the child, particularly of the sort of child to whom Dr. Davis referred, who seems obviously to have a seriously impaired auditory system but about who one must always ask the question: is the interference cortical, aphasic or dysphasic, or something of that sort, rather than related solely to end organ? We have not gone very far yet—by "we" I should include the entire range of knowledge in this phenomenon-analysis—toward dissociating or dissecting out the various factors of the behavioral complex.

LXXI

A REVIEW OF MÉNIÈRE'S ORIGINAL PAPERS IN THE LIGHT OF OUR PRESENT KNOWLEDGE OF MÉNIÈRE'S DISEASE

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Few medical writers have been more widely quoted than Prosper Ménière, and few can have suffered more from a seeming reluctance on the part of otologists to read or, if having read, to get the sense of, his papers.

It appears to be the practice everywhere, both in textbooks and papers, to start the exchange of opinion in regard to this disturbance with the statement that Ménière believed that hemorrhage into the labyrinth was the cause of the group of symptoms he described. Some go further and state that he put forward as the pathologic basis of this disease a necropsy report concerning a girl who died of some condition which produced the symptoms of vertigo, vomiting and deafness, but that whatever this patient had, it obviously was not Ménière's disease.

In reading Ménière's original papers,[1-5] I found both these statements to be contrary to the facts.

In the year 1861, the last year of his life, P. Ménière, assistant professor of the faculty and physician to the Imperial Institute for Deaf Mutes, presented a group of papers[1-5] in the *Gazette médicale de Paris,* culminating in a final paper,[5] the one usually quoted, which was published on September 21, 1861, entitled "Mémoire sur des lésions de l'oreille interne donnant lieu à des symptômes de congestion cérébrale apoplectiforme." In the introduction to this paper he stated, "There has been presented to my observation over a long period a number of patients showing a group of symptoms invariably the same, symptoms of a grave appearance, giving the impression of an organic lesion of the most serious type, recurring from time to time during weeks, months or years, disappearing suddenly and leaving as the usual result the complete abolition of a sense. Permit me the description of one of these pathologic conditions which everyone has encountered, and of which you should realize the importance

I have attached to it because of the symptoms and signs which accompany it and because of the infirmity which is the consequence of it."

At the time Ménière wrote, physicians were lumping together all cases which were characterized by vertigo, nausea and vomiting under the diagnosis of "apoplectiform cerebral congestion," and were treating the unfortunate persons who presented such symptoms by means of copious bloodletting and violent purgation, a treatment which Ménière stated the patients were fortunate to recover from in some months.

Ménière said, however, "Vertigo, nausea and vomiting are but the symptoms of disease, and what the disease is ought to be determined before hazarding a treatment which may harm the patient." Further, "It is possible to attach certain of these symptoms to a specific cause, to attribute vertigo and falling to lesions different from those which have their site exclusively in the brain, and as a consequence to institute a rational treatment to these affections, for too long a time confused under a single title."

He felt that it was possible to separate from the general group of patients having vertigo, nausea and vomiting as the presenting symptoms, a number of persons who had symptoms and signs which suggested that, in addition, cochlear involvement was present. Ménière stated, "Attacks of sudden vertigo, nausea and vomiting are quite common. I have seen a large number of them. There is, however, a perfectly distinct group of such patients, the specific nature of which is characterized by loss of auditory sensitivity . . . If those who might have the occasion to observe the sudden onset of vertigo, nausea and vomiting in their patients would take the pains to examine carefully the auditory apparatus, if they measured the degree of hearing in both ears, if they would question their patients as to what had taken place in these organs since the appearance of these 'congestive accidents of the brain,' they would recognize that the inner ear is the site of the lesion, the point of origin of the symptoms which had previously appeared so dangerous to life, and against which they had employed a rigor of treatment as uncomfortable to the patient as it was futile, a violent therapy of which not the least important inconvenience is not only to harm the patient but also to cause him to doubt the intelligence of his physician."

Ménière described the deafness present as a nerve type, with greater inability to hear bass voices than treble. He stated that the disturbance might involve both ears, but that more commonly one was involved. During what he called "the crisis" of the disorder he observed that the patient tended to lie on the sound ear, and

might give the impression of having bilateral deafness unless the head was turned during the examination. He pointed out that examination of the external auditory canal, the ear drum and the middle ear showed no evidence of disease, and that previous to the onset of the disease the patient had had no symptoms in regard to the ears. He stated that a bruit in the involved ear or ears was characteristic of the condition, in that it could not be interpreted by carotid pressure, thus indicating its neural origin, and in that it could be continuous or intermittent but continued between attacks. Both the tinnitus and the recurring episodes of vertigo, nausea and vomiting stopped when the hearing in the affected ear or ears was completely lost, a loss which might not occur, however, until some years had passed.

Ménière described four types of vertigo and dizziness in the "crisis" or "attack," and one type present between attacks. First, the patient might have a true rotatory vertigo, coming on without warning and tending toward remission and recurrence. The patient who has this type of vertigo will, if lying in bed, on opening the eyes see the room whirling around him, and attempts to move will produce violent nausea and vomiting. Ménière stated that the duration of the attack was from two to twenty-four hours, and that during the attack the patient does not lose consciousness, has no weakness or paralysis, can use the tongue freely, and on termination of the attack is in perfect health and can describe the events of the illness without difficulty.

Second, he described a condition in which the patient both feels and is unsteady. On attempting to walk he might veer to one side or the other and bump into a stone wall or a tree.

Third, the patient might experience a to-and-fro or up-and-down motion. One of Ménière's patients described his sensation as that of "being on the bridge of a vessel at the mercy of a stormy sea."

Fourth, the patient without previous warning might be thrown violently to the ground as though struck on the head, and afterward, while lying down, experience violent vertigo, nausea and vomiting.

Fifth, the patient between "crises" is unable to turn the head suddenly, or to lie down in or get up from bed without experiencing some degree of either vertigo or dizziness, although rarely to a severe degree.

Ménière also described a syncopal state with pallor, cold sweat and feeble pulse as being associated with the severe attacks of "crises."

Crowe[6] said that the symptoms Ménière described were so invariable that they must originate from a single definite cause, and the disturbance therefore warranted the term "disease" being applied to it.

Little has been added to Ménière's original description of the disease. Both Crowe and Mygind and Dederding[7] added the important fact that the hearing loss in Ménière's disease is variable, and both Dederding[8] and Lillie, Horton and Thornell[9] noted that the deafness is reversible under treatment. The fact that nystagmus of the third degree is always present with vertigo of any severity was noted by Brunner.[10] Other than these few additions, Ménière's original description appears to have been complete.

He also noted that in injuries to the ear drum, vertigo is not produced unless the ossicular chain is involved, and he also observed that inflation of the ears might produce vertigo if the ear drum is intact, but that if the ear drum is perforated, vertigo will not ensue. He felt that this demonstrated that some movement of the ossicles which forces the stapes into the oval window is necessary for the production of vertigo. This fact, he felt, demonstrated that vertigo can be produced by irritation of the equilibrial labyrinth. It can be noted that Ménière recognized that some degree of unsteadiness or dizziness might be produced by occlusion of the eustachian tube, but he apparently had not observed any patients in whom such occlusion had produced the group of symptoms he described.

Ménière also called attention to the fact that medication with quinine will make the condition he described worse, and he reported a case in which this disorder occurred directly after medication with large doses of quinine taken for a fever.

When it is realized that Ménière described attacks of vertigo recurring frequently during weeks, months or years, it becomes hard to understand why the hypothesis that hemorrhage into the labyrinth is the cause of this disturbance should have been attributed to him.

After considering his observations, I think the statement would be justified that Ménière was an astute clinical observer with excellent powers of analysis and a clear literary style, and that he definitely described what was then a new clinical entity.

In 1924, however, in reviewing his work, M'Kenzie[11] stated that there was little of the French love for order and arrangement in Ménière's style. He further said that the myth of Ménière's original case had insinuated itself so stealthily into otologic literature that we are all in the habit of looking on its traditional data as proved. He felt that it is well to refer occasionally to original sources, and

for this reason he had carefully reviewed Ménière's original work stating, "The result of my efforts was a surprise."

M'Kenzie accused Ménière of giving from an imperfect memory the pathologic findings in regard to a young woman whose condition he had first described thirteen years previously (1848). He implied, as Dederding[8] and Atkinson[12] subsequently did, that Ménière presented this case as demonstrating the pathologic basis for his disease. M'Kenzie showed, it is true, that in Ménière's original account of this case he stated that the entire labyrinth, rather than the semicircular canals alone, was filled with reddened plastic lymph. Ménière may have made this omission to strengthen the hypothesis he was presenting: that a lesion of some sort in the semicircular canals is the cause of the recurring symptoms of nausea, vomiting and deafness with tinnitus. If this were so, the small stratagem hardly seems to warrant the harsh criticism to which it has subjected him, especially when it is realized that the original account would have given a better explanation of the combination of cochlear and equilibrial symptoms than the later one he presented.

M'Kenzie appeared annoyed with Ménière because there was no evidence that Ménière had made the statement, repeated so often in our textbooks and papers, that he called the lesion in the labyrinth a "hemorrhage" or an "apoplexy." M'Kenzie was unable to discover where this almost universal misquotation originated.

When the context of Ménière's paper is read before and after the description of the famous case, it is obvious that he had no intention of presenting this case as typifying the pathologic background of the symptom complex which he had described. I have already shown that Ménière was intent on demonstrating to his confrères that vertigo, nausea and vomiting need not necessarily rise from "lesions of the cerebellum or the cerebellar peduncles," but that when these symptoms are accompanied by deafness the site of the disease lies in the inner ear. To illustrate this point he presented a case in which a young girl was suddenly attacked by vertigo, nausea and vomiting and sudden complete deafness. Ménière had seen many instances of "Ménière's disease" and had pointed out the favorable prognosis of life with which the condition is associated. It is clear that he knew as well as do his modern detractors that patients never die directly from this disturbance, but only secondarily. It seems unreasonable, therefore, that he should have been charged with the attempt to present this case as the basis of the specific group of symptoms he described.

This error of comprehension is readily demonstrated by a translation of the necropsy report given by Ménière, together with a portion of the context before and after.

"In order that we may attain to some kind of clear knowledge on the subject, I give a literal translation of Ménière's account.[11]

" 'Some patients more attentive to that which goes on in themselves, have made it possible for me, with the help of very precise questions, to establish that vertigo, the syncopal state, the sudden falling of the body, vomiting, have been preceded by noises in the ears, that these noises do not show any appreciable cause, that they persist in the intervals between attacks, but that they often coincide with an increase in the deafness, and that these noises never take a pulsating arterial form; in a word, they do not originate from the carotid artery. That is an indication of their nervous character; they arise from a particular state of the acoustic nerves, and not from any cause connected with the blood; the circulatory system has nothing to do with them. What is it, then, that manifests itself in these other kinds of symptoms, noises which persist with a remarkable obstinacy, the hearing more and more enfeebled and, as I have been able to note, its complete abolition in a case where the ear had not been the site of any infection? . . . I am not able to forget that beyond the drum, there lies an apparatus which is so mysterious that it has not revealed to us all the phenomena which go on there. The inner ear has revealed to us several of the secrets of its organization, pathologic anatomy has demonstrated that certain forms of deafness are due to tissue alterations which one is able to recognize, and finally, experimental physiology has subjected some of its parts to experiments capable of throwing light on the nature of certain functional troubles. I have spoken before, a long time ago, of a young girl who, having traveled by night in winter on the top of a stage coach, when she was at a catamenial period, had in consequence of a considerable chilling, a *complete and sudden deafness.* She manifested as her chief symptom continual vertigo, the slightest effort to move produced vomiting, and death followed on the fifth day. *The necropsy showed that the cerebrum, cerebellum and spinal cord were completely free from any alteration,* but as the patient had become suddenly deaf after always having had perfect hearing, I removed the temporals in order to examine with care what could be the cause of this complete deafness, so rapidly coming on. *The sole lesion I found was the semicircular canals filled with a red plastic material,* a sort of blood-tinged exudate of which scarcely any traces were perceived in the vestibule, and which did not exist in the cochlea. May one, on the authority of a single fact, establish the necessary correlation between vertigo, deafness and a lesion of the semicircular canals? We would not have the temerity to reply affirmatively to this question if nothing came to the help of the observation; but in the presence of the experiments of M. Flourens[13] on sectioning of these canals, and taking into account the alterations of function which followed these artificial lesions and are present in all animals, one is brought to think that the symptoms which appear in man and which consist of vertigo, nausea, the syncopal state, which is accompanied by ear noises, and which has deafness as its consequence, may depend on an alteration which has as its site that portion of the labyrinth of which we have spoken.' [Italics not in original.]"

I believe that the quotation establishes unequivocally that Ménière's only purpose in presenting this case was to demonstrate that a lesion in the labyrinth could produce deafness, vertigo and vomiting. There is no evidence to indicate that he intended to imply that the case demonstrated the pathologic background in the

particular group of patients he had described. Indeed, he was so far from doing this that in regard to the type of lesion causing Ménière's disease, he recalled that he had seen individuals with hemicrania who exhibited phenomena analogous to those he had found present in the disturbance he described. He found some types of migraine associated with tinnitus, vertigo and a gradual loss of hearing which resisted all forms of treatment. Ménière even went so far as to state that he believed that microscopic researches on the labyrinth would reinforce his opinion that the disturbance had its origin there and would *throw some light on the nature* of the condition he described. He felt, however, that he had presented sufficient evidence to establish the fact that the lesion causing the symptoms he had described lay in the labyrinth. With the means at his disposal, however, he was unable to demonstrate it pathologically. In his résumé Ménière stated, "These functional troubles having their site in the inner ear are capable of producing symptoms attributed to the brain . . . Everything leads to the *assumption* that the lesion which is the cause of these functional alterations lies in the semicircular canals."

Both M'Kenzie and Atkinson also called attention to the fact that Ménière had failed to mention the symptom of vertigo in the first account of this case. Atkinson stated, "In the earlier account of 1840, [sic] there is no mention made of the vertigo which is made so much of in the later one. . . The reason for this discrepancy is difficult to assign. Was his memory at fault or did he through carelessness omit any description of vertigo in his first account?" Atkinson felt that the explanation of "carelessness" was the most probable one, "For he was a fluent and prolific writer and not very careful even of his grammar . . . There was none of the French precision about Ménière's writing, though his clinical observation was accurate enough . . . Or did he unconsciously embroider fact to conform to theory; did he remembering the pathologic findings in the case which fitted so well with his idea 'remember' also the vertigo?"

It seems rather strange, however, that clinicians of such wide experience both should have wondered if vertigo had really been present in the patient discussed, since they produced no evidence to discredit the impression that the patient had some sort of condition producing acute destruction of the labyrinth.

When Ménière's translation of Kramer[14] is read, no difficulty is presented in understanding why no mention of vertigo or vomiting was made in Ménière's first account of "the case." This account occurs in the "additions du traducteur" which Ménière made at the

end of chapter III, which is entitled "Maladies de l'oreille interne."
Ménière began by saying, "The third chapter of the work of Kramer
leaves much to be desired. It contains the details of but a single
type of disease, nerve deafness, and it appears impossible to me to
agree with his views." He goes on to urge that until the changes
which take place in the structure of the organ are known, it will
not be possible to integrate symptoms and disease. He stated:

"It is sufficient to meet with one well-marked case, to recall the
principal circumstances of it, and to make a careful dissection of
the diseased petrous to establish with sufficient precision the corre-
lation between the type of lesion and the symptoms observed during
life, and this having been done it will put us on the trail of other
analogous diseases.

"I have seen for instance a young girl suddenly struck with
complete absolute deafness in the short space of several hours. Trav-
eling in an open carriage she was exposed during the night to a
severe degree of cold, during her menses, and the hearing was lost
without the ears having been the site of pain. Death, which prompt-
ly followed, permitted me to dissect the temporal bones with care,
and I found in the whole labyrinth a kind of reddened plastic
lymph, which appeared to be the product of a secretion of all the
surface membranes lining the internal ear. In a very similar case,
but one in which death occurred much later (two months after
sudden loss of hearing), I found the same plastic lymph of a clear
yellow color, peppered with a multitude of small opaque gray points,
closely resembling beginning tuberculous granulations."

Here again, when Ménière's report of this case is read in its
original context, it would seem probable that he did not mention
vertigo and vomiting because there would seem no reason for him
to have done so, since the entire discussion was about the possible
causes of perceptive deafness.

In both instances, the translators of Ménière's paper seem to
evidence a sort of perverse desire to misunderstand. This cannot be
laid to difficulties with the French language, because both authors
qualify themselves as experts in the field, by adverse comments on
Ménière's grammar, by a curious coincidence using nearly the identi-
cal words. My French is not sufficiently good to enable me to verify
their observations as to Ménière's grammar. Wells,[15] however, stat-
ed, "He clearly was a man of culture and wide interests; a physician
who possessed not only skill but learning; not only learning but abil-
ity; and not only ability but character."

The pathologic aspects of the condition were only recently (1938) demonstrated when Hallpike and Cairns[16] were able to present the histologic findings in two authenticated cases of Ménière's disease in which the patients succumbed to hemorrhage into the cerebellum after intracranial division of the eighth cranial nerve. Their finding, that of primary dilatation of the ductus cochlearis with secondary dilatation of the utricle and saccule, with the conspicuous absence of any inflammatory change, frequently has been confirmed, and now is well known to all.

COMMENT AND CONCLUSIONS

When Ménière's papers are read, the reader is forced to conclude that Ménière had a better insight into the disturbance now named after him than many of his otologic descendants have had. They answered his plea for better differential diagnosis by substituting for "apoplectiform cerebral congestion" a similar catch-all, "Ménière's syndrome" or "symptom complex," which they used to avoid the mental fatigue of differential diagnosis. In answer to his plea to take primary note of the distinguishing cochlear changes, they have established vertigo as the "cardinal symptom" of Ménière's disease. To his plea to study the inner ear microscopically, so as to discover the cause of the disturbance, they gave no heed until a neurosurgeon recently furnished them the necessary impetus. It is not a record that redounds to the credit of otology, but it makes the original description of Ménière's disease seem the more prodigious by comparison.

Ménière undoubtedly deserves the honor of having the disease entity he described named after him, not only because he was the first to describe it, but also because in these papers he was the first physician to apply to clinical medicine the newer knowledge gained by the physiologist and to start medicine back from that preoccupation with the dead house into which it had been forced by Virchow and his followers.

MAYO CLINIC.

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LXXII

POSTMENINGITIC COMPLICATIONS WITH PARTICULAR REFERENCE TO OTOLOGIC SEQUELAE

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To the epidemiologist, meningococcus meningitis presents the problems of prevention, detection and treatment of the disease. However, it is obvious that the effects of meningococcus meningitis may extend beyond the period of convalescence from the acute phase of this illness.

During the past 15 years, patients who were discharged from South View Hospital have occasionally returned because of complaints during or, at times, long after the convalescence from meningococcus meningitis. In most instances the chief complaints have been headache, loss of hearing, or hearing defects, muscle pain, muscle twitching, fainting spells, and rarely, convulsions. With the decreasing mortality and morbidity rate made possible by the recent advances in therapy, it is probable that more cases of postmeningitic sequelae will be observed. Our own study of the recovery rate revealed that during the years 1928 and 1929, when the patients were treated with polyvalent antimeningococcus serum alone our mortality rate was 19%, and that in 1944, with the advent of the sulfonamides and penicillin, our mortality rate dropped to 8.8%, a reduction of more than 50%.

CLINICAL STUDY

Because of our belief that sequelae following this disease will assume a position of increasing importance in the years to come, we undertook a survey of 323 cases of meningococcus meningitis which we treated during the ten-year period of 1936 to 1945 inclusive.

1. *Mortality and Morbidity*: These cases are analyzed from
the standpoint of morbidity and mortality in Table 1 and Table
2. There were 55 deaths, a mortality of 17.7%. Table 1 indicates
the mortality rate for the various age groups and Table 2 for the
various years. Table 2 shows that the cases of meningococcus men-
ingitis for the years 1936 to 1942 inclusive might be classified as
sporadic, but during the years 1943, 1944 and 1945 they reached
epidemic proportions.

Smithburn[1] and his group studied a series of 144 cases at the
Indianapolis City Hospital occurring during the period from No-
vember 11, 1929, to April 1, 1930. They reported 92 deaths which
represented a 67% mortality. Neal[2] and her group reported a mor-
tality of nearly 30% for 627 cases and pointed out that the mortal-
ity rate varied from year to year. McLean and Caffey[3] reported
on 44 cases of sporadic meningococcus meningitis, giving a mortality
rate of 50% for infants under one year of age and only 15% for
children from one to five years of age. During an epidemic in the
years 1905, 1906 and 1907, Cohn[4] studied 82 cases in which only
32 patients recovered, a mortality rate of 61%.

The statement of Smithburn[1] and his co-workers that the
mortality rate varies from year to year is corroborated in our own
series in Table 2. It may be noted that all the patients admitted dur-
ing the year 1938 died, whereas there were no deaths in the year
1940. McLean and Caffey's experience[3] of 50% mortality in infants
under one year of age and only 15% for children between the ages
of one and five years is not borne out by our own statistics which
show only a 12.5% mortality for infants and 21.7% mortality in
children between one and five years of age. The marked difference
in these statistics is probably explainable by a varying virulence of
the disease from year to year.

Antibiotic drugs are apparently more effective than polyvalent
antimeningococcus serum. It may be noted that prior to 1939 we
had 17 cases and 7 deaths, a mortality of 41.1%. From 1939 to
1945 inclusive, in which years antibiotic drugs were used, we report
316 cases and 48 deaths, a mortality of 15.2%. Cook[5] and his co-
workers showed a survival in 36 of 87 cases or a mortality of 70.6%
in a report of an epidemic in Cincinnati in 1930 and 1931. It
would therefore seem that the use of antibiotic drugs does play
some part in the reduced mortality of recent years in spite of a pos-
sible variation in virulence from year to year.

2. *Study of Sequelae*: Since we were chiefly interested in all
types of sequelae, our first aproach to this study was in the nature

of a questionnaire which was mailed to each of the 268 survivors. In the questionnaire we first explained the importance of our aims and then, through a series of carefully worded questions, we inquired in essence as to the presence of sequelae, their types and nature. From this questionnaire and our hospital records we were able to assemble certain significant data.

From the 268 survivors to whom questionnaires were sent we received 178 returns, 88, or 49.4%, of which gave a history of some residual disorder, whereas 90, or 50.5%, felt that they had suffered no residual effects (Table 3). The duration of these sequelae is somewhat variable. Sixteen of the patients became free of symptoms; this is considerably higher than the incidence reported in 1926 by Neal, Jackson and Appelbaum[2] of about 18% in a group of 650 patients. Top,[6] in his monograph on communicable diseases, reports a five-year follow-up study of 100 cases of meningococcus meningitis and reports an incidence of 25% of severe disabilities resulting from this disease. Degen,[7] in his survey of 986 patients, found that 387 (39.2%) complained of disabilities.

In compiling our statistics, we, of course, are not unmindful of the possibility that at least some of the sequelae reported by our patients may have been defects which pre-existed and for which the disease was blamed.

The types and incidence of sequelae resulting from acute meningitis are tabulated in Table 4. It is of interest to note that headache was the chief residual complaint. The headache was most often occipital in location but varied considerably in distribution. Cord symptoms, represented by muscular and joint pains as well as muscular twitching were the next most common sequelae. Fainting and convulsions were rather uncommon. A rather high percentage of patients described hearing defects. Almost 13% noted this disability within a relatively short time after the acute phase (Table 4). Table 5 represents the residuals which remained a year or more after the acute illness. All but one of the 23 patients who described hearing defects had these defects after a year, a total of 12.3%. In Degen's study,[7] a total of 8.3% of the patients complained of hearing defects.

3. *Otitic Sequelae*: Because of our special interest in these hearing defects a follow-up questionnaire was sent to the 22 individuals who mentioned this persistent disability in the original questionnaire. In this follow-up letter we asked the following questions:

1. Did difficulty in hearing start with the onset of meningitis?
   Yes. No.
2. Do you still have an impairment of hearing? Yes. No.
   Which ear? Right. Left. Both.
3. Has there been any improvement? Yes. No. How?
4. Is the deafness worse in one ear than the other? Yes. No.
   Which ear? Right. Left.
5. Are you wearing a hearing aid? Yes. No.
   Does it help you? Yes. No.
6. Does your deafness change with the weather? Yes. No.
   How? When?
7. Do you hear better over the telephone? In a noisy room?
   When you can see the face of the person speaking?
8. Have you consulted a doctor about your hearing? Yes. No.
   With what results?

Of the 22 patients who were sent this questionnaire 14 indi-
cated that their hearing impairment was still present. One was
excluded from our clinical examination because his hearing defect
antedated his illness and was not increased thereby. During the
year 1948, 11 of the remaining patients reported to one of us
(Grove) for a clinical examination of hearing function.

Considering these 11 patients critically, it was found that 3
had a total bilateral deafness. Four revealed a unilateral total loss
of hearing with residual hearing present in the other ear. Of these
4 the residual hearing of the opposite ear was normal in 2, moder-
ately impaired in 1, and markedly deficient in 1. In a third group
of 4 patients, 2 had unilateral incomplete hearing impairment while
the opposite ear was normal. In the remaining 2 cases the hearing
defect was bilateral and considerable (Table 6).

These 11 cases were examined audiometrically for air and bone
conduction, with tuning forks and by voice tests, monitored and
unmonitored. In the 8 cases with a measurable hearing function,
the impairment was of the mixed type in 7 and of the perception
type in one. This is at variance with the findings of Johnsen,[8] who
found a perception type of hearing loss in all of his cases.

Thirteen of the patients answering our questionnaire reported
that their hearing impairment continued unchanged (Table 7), al-
though 6 indicated that they had noted some gradual improvement.
Six noted a variation of their hearing impairment with weather
changes while 8 did not. Two reported benefit from the use of a
hearing aid. Eleven did not use an aid.

A control series of 14 patients was recently examined clinically.
None of them claimed any hearing loss as the result of their men-

ingitis. In 9 of these patients the hearing was found to be entirely normal. In the remaining 5 patients, aged 38, 55, 58, 68 and 71 years, such hearing loss as was present, and which involved only the higher frequencies, could very well be ascribed to presbycusis.

In 1918, Versteegh[9] found a pronounced deafness in 6 out of 48 cases, or 12.5%. However, in 19 of the 48 patients, who were unaware of any hearing impairment as the result of their illness, he found deviations from the normal consisting for the most part of islands in the middle range. Smithburn[1] and his co-workers found 11 with impaired hearing among 92 survivors, or 12.5%. Cohn[4] reported 3 cases of deafness in 27 survivors of an epidemic of pre-sulfa days, an incidence of 11.1%. The incidence of hearing impairment reported by Neal[2] and her group was 7.7%. McLean and Caffey[3] reported an incidence of 9% and Cook[5] and his co-workers, 5.5%.

The classic report of Shambaugh, Hayden, Hagens and Watkins[10] on "Children in Public Schools for the Deaf," which involved a study of 5,348 children, indicated that 385 had acquired their deafness as a result of epidemic meningitis. Of these, 230 were apparently totally deaf while 155 had some vestiges of hearing. Two hundred and sixty-eight of these children exhibited no evidence of vestibular disorder.

Politzer[11] in his textbook states that the intensity of the disease has less influence on the development of deafness than its epidemic character. He states that while in very severe cases of meningitis there is often recovery without any hearing impairment, complete deafness may occur in abortive forms. He cites Gottstein as having observed complete deafness in patients who complained only of exhaustion, headaches, and neck rigidity for a few days. Politzer[11] is also the author of the statement that in some epidemics only a few patients lose their hearing while in other epidemics the majority of those who survive become deaf. This statement is concurred by Johnsen.[8]

According to Birkholz,[12] it is difficult to postulate the exact time of the onset of the deafness because of the overwhelming coma and somnolence of the acute state. Politzer[11] believes that the hearing impairment occurs in the first two weeks of the illness, but Johnsen[8] states that in the days before chemotherapy it sometimes manifested itself late in the disease or during an acute exacerbation. In 2 of Cohn's[4] 3 cases the deafness came on in the fourth week. Smithburn[1] reports that the hearing defect appeared in 3 cases during the second week, in 2 during the fourth week, and in 1 case during the
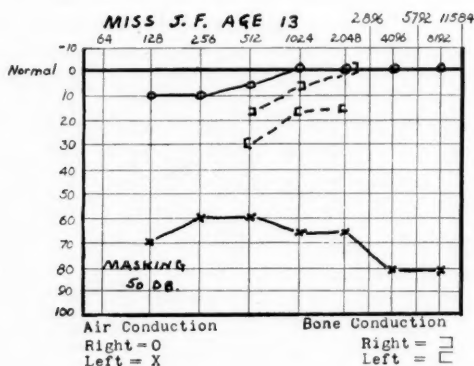
Fig. 1.—This audiogram represents a marked hearing loss in one ear with retention of fairly normal hearing in the opposite ear.

fifth week. Moos[13] reports 15 cases in which the hearing defect first appeared in the period between two weeks and four months.

In 7 of our cases the hearing impairment was bilateral and in 4 unilateral. This is in accord with the experiences of Politzer[11] and Johnsen[8] but does not coincide with the experience of Smithburn[1] and his group who found only 2 instances of bilateral loss in 11 cases of hearing impairment.

In 10 of the 22 ears examined by us, the hearing loss was total and complete. In the other 12 ears, the defect was partial in 8 and in 4 ears the hearing was normal. The patients described by Ryland,[14] Davis,[15] and Cohn[4] were all completely deaf bilaterally and of the 26 patients reported by Neal, Jackson and Appelbaum,[2] 21 were totally deaf.

Summarizing our own series of 11 patients: 3 were totally and bilaterally deaf, 2 exhibited total deafness in one ear and entirely normal hearing in the other; in 2 the hearing was normal in one ear and deficient in the other ear; 2 had a total loss of hearing in one ear and considerable impairment in the other; while 2 had a bilateral impairment of hearing but could carry on a normal conversation.

Figures 1, 2 and 3 illustrate representative cases from our series.

Johnsen[8] states that vestibular disturbances are not particularly conspicuous, being usually masked by the general symptom complex of the meningitis and the general debility of the patient. None of
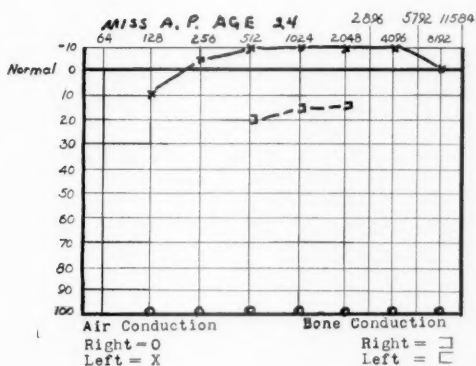
Fig. 2.—This audiogram represents total deafness of one ear with normal hearing in the opposite ear.

the patients examined by him after the lapse of several years exhibited either nystagmus or a positive Romberg test. On the other hand, Politzer[11] found vestibular disturbances in two-thirds of his own patients and cites Moos as having found them in 50% of his patients. He states, however, that these vestibular disturbances usually disappear more rapidly in older than in younger patients. In two of Cohn's[4] cases the caloric irritability was absent after two years. Ryland[14] and Davis[15] found complete loss of caloric irritability.

The general consensus in the literature seems to be that the vestibular branch of the eighth nerve suffers as frequently as does the cochlear branch. Only 2 of the 11 patients which we recently examined complained of any dizziness. This was of minor degree, was not in any way incapacitating, and was not associated with any other disturbances of equilibrium or co-ordination. As considerable time had elapsed since their hospitalization we did not deem it advisable to investigate this phase except by means of the history. We are not prepared at this time to offer any opinion as to the rate of occurrence of vestibular disturbances in our patients or as to the permanency of such disturbances. If, however, our concept of the pathology which causes the deafness is correct, we believe that the vestibular branch of the eighth nerve must suffer equally with the cochlear branch.

Brunner[16] believes that the pathologic basis for the perception deafness is either retrolabyrinthine neuritis acoustica with hyperemia
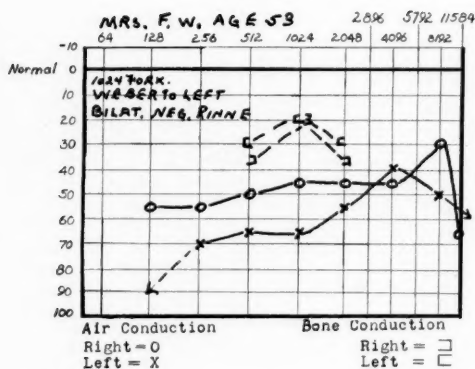
Fig. 3.—This audiogram represents bilateral loss of hearing follow-
ing meningococcic meningitis.

and cellular infiltration between the brain stem and the internal
auditory meatus, or probably more commonly, an injury to the
sensory end organ.   According to him, the toxic or bacterial pro-
ducts reach the labyrinth through preferred spaces, the perineural
sheaths, or the cochlear aqueduct.   Wittmaack[17] states that a serous
or serofibrinous exudate first appears in the cavity or membranous
parts of the labyrinth followed by cellular infiltration.   Connective
tissue formation or suppuration of the labyrinth follows.

In 1881, Moos, cited by Benesi,[18] described the nerves in the
internal auditory meatus as showing first a hyperemia and cellular
infiltration.   Later they were bathed in pus.   In 1886 Steinbrugge,
cited by Benesi,[18] found histologically that the nerve fibers in the
internal auditory meatus were bathed in pus.   There was blood ex-
travasation and extension of the infiltration into the modiolus and,
in one case, the nerve fibers had disintegrated to a considerable de-
gree.   In 1890, Schultze, cited by Benesi,[18] demonstrated atrophic
sclerotic changes in the eighth nerve in one case.

In a table of the histologic findings in 20 cases listed by Haber-
man, Lucae, Steinbrugge, Larsen, Nager, Borke, Alt, Agazzi and
Hellman, Birkholz[12] gave the following pathologic picture:

1.   The labyrinth showed purulent involvement in 17 cases.

2.   The eighth nerve showed neuritis or perineuritis in 19 cases.

3.   The aquaeductus cochleae showed pus or evidence of inflam-
mation in 8 cases.

4. The aquaeductus vestibuli showed pus or evidence of infection in 6 cases.

The organ of hearing can be damaged intracranially between the brain stem and the porus acousticus or in the labyrinth. As there is no supportive evidence that meningococcus meningitis causes encephalitis, we must discount damage to the cochlear nucleus or the center of hearing. Damage to other cranial nerves by the basilar meningitis has been reported by Smithburn and others, namely, facial paralysis, ocular palsies, glossopharyngeal palsies, and spinal accessory palsies. As a rule these clear up completely. The deafness does not. While we cannot rule out damage to the eighth nerve between the brain stem and the internal auditory meatus in some cases, we believe that in the majority of the cases the damage to the hearing function takes place in the labyrinth.

While we agree in general with the views of Brunner, Wittmaack, Moos, Steinbrugge, Schultze and Birkholz, that the pathologic factor causing the deafness or impaired hearing in meningococcus meningitis is an actual infection of the eighth nerve or a purulent invasion of the labyrinth, we cannot dismiss without a word the contention of Leichinger and Abelson[19] that it may be due to an exotoxin circulating in the blood. They base their idea upon one case of meningococcemia without any symptoms of meningitis in which tinnitus and a hearing defect, which had not been present on admission, appeared on the third day of the disease. They contend that, since no meningitis as evidenced by meningeal signs was present in this case, the deafness could not be attributed to an infection or purulent invasion of the eighth nerve or the labyrinth. They compare the deafness in this case to that occurring in mumps, diphtheria, scarlet fever and tetanus, in all of which, with the exception of mumps, an exotoxin is elaborated. They support their opinion with the experience of Beck,[20] who demonstrated definite changes in the cochlea and spinal ganglion of rabbits after the intravenous injection of Bacillus diphtheriae, staphylococcus, and Bacillus dysenteriae. In this connection they quote the work of Ferry, Norton and Steele,[21] who demonstrated that the meningococcus can produce an exotoxin.

We do not believe that Leichinger and Abelson[19] have entirely excluded the presence of a mild meningitis in their case by simply stating that there were no meningeal signs present. The preponderance of pathologic evidence produced to date seems to indicate that the deafness and hearing impairment which follows meningococcus meningitis is due to an actual infection or a purulent invasion of the eighth nerve or labyrinth or both.

As to the prognosis of postmeningitic deafness, Politzer[11] states that "cases of deafness are but seldom met with in which the power of hearing in one ear, rarely in both, undergoes such improvement in convalescence or later, that speech can be understood even at a short distance." And again, "the return of the hearing power for speech is not always permanent, as the author's experience has shown that complete deafness may return again at a later date." Cook[5] and his co-workers reported a partial deafness in two cases which became complete seven months later. According to Birkholz,[12] patients with slight hearing loss may improve somewhat but the hearing defect is always demonstrable, and those with pronounced impairment rarely improve. Cohn[4] states that while most of the other paralyses caused by meningococcus meningitis recover, the most dreaded complication, deafness, always remains permanent. Smithburn[1] and his co-workers state that the deafness in their 11 cases did not disappear and in most of them was not improved. In a small number of the cases reported by Neal[2] and her group there was partial or complete recovery from the deafness.

In general we agree with the majority of the authors that the hearing impairment caused by meningococcus meningitis is usually permanent and sometimes progressive. In answer to our questionnaire, 6% noted some subjective improvement in hearing after a lapse of time.

PATHOLOGIC PHYSIOLOGY

After surveying the general sequelae of this disease it is quite evident that some transformation of the leptomeninx must have taken place as a result of the acute purulent inflammatory stage. As the infection comes under control, the extensive purulent exudate becomes organized. Organization is brought about by gross proliferation of fibroblasts and the formation of a considerable amount of fibrous scar tissue. This process is responsible for the occlusion of pathways in the leptomeninx and subsequent interference with the free circulation of cerebrospinal fluid. This course of events not only occurs on the vertex, base of the brain, and medullary areas, but also throughout the length of the spinal cord and along the nerve roots. The acute exudative phase of the disease plus the subsequent adhesive fibrosis of the healing phase leaves the surviving patient with a central nervous system often damaged to varying degrees. Bailey[22] makes mention of infantile hemiplegia, and diplegia as well as paraplegia in the adults, deafness, deaf mutism, aphasic difficulties, and perhaps what is the most common complication, chronic hydrocephalus. Adhesive fibrosis in itself does not seem to offer the only explanation for the occurrence of sequelae, for on

careful evaluation of these cases, it is quite evident that there must be circulatory impairment to the brain and spinal cord as well. At the onset of the infection, there is no restraining force on the amount of fibroblastic proliferation. One cannot conceive of an extensive fibroblastic organization of the exudate which would not cause some residual interference with normal neurophysiology. The result of this fibroblastic process is a permanent cicatrix to brain and cord, and quite naturally, interference with normal function. Necropsies performed on a small series of cases and reported by Bailey[22] disclosed this particular aspect to have occurred in the acute stages of cerebrospinal meningitis. The nerve roots including those of the cranial nerves may readily develop a fibrous perineuritis. We feel that most of the sequelae which occur may be traced directly to these pathologic changes.

However, if we were to accept these rather static fibrous changes of the leptomeninx as the sole cause of the sequelae, we would be hard put to explain the variability of symptoms produced by such factors as changes in weather and occupation (Table 8). We must, therefore, look to the alterations in dynamics produced by the insult to the central nervous system. The hot weather headaches and occasional convulsions may be attributable to waterlogging of the central nervous system, resulting in a strain or distortion at the point of adhesion. The interchange of water between the three main components of the body, the vascular system, the cells, and the extracellular spaces, is dependent upon such factors as osmosis, hydrostatic pressure, electrolyte concentrations, vascular permeability, and probably other less well recognized factors. Water intoxication can occur in human beings following the ingestion of large amounts of water combined with a loss of salt.

How such factors as water balance affect the brain of these postmeningitis patients can only be speculated upon in the present state of our knowledge of the subject.

CONCLUSIONS

From our ten-year survey of 178 cases of meningococcus meningitis, and correlated with the experience of others, we conclude that the prognosis as to life is becoming better since the advent of chemotherapeutic and antibiotic drugs. It would seem that as a result of a lowered death rate we may anticipate seeing a larger number of survivors who complain of sequelae. Chief among these complaints are those relative to a damaged nervous system and disturbances in hearing. The variability of constitutional symptoms does not appear to be a static thing in over a quarter of the cases, indi-

cating that normal neurophysiology is often inconstantly altered by the pathologic changes which occur.

From a critical examination of 11 patients with post-meningococcus meningitis who exhibited hearing defects, and from the results reported by others, we feel that the hearing defect caused by meningococcus meningitis is usually permanent and sometimes progressive.

We further believe that the variability of symptoms occurring in a postmeningitis patient can be explained by abnormal physiological changes produced by a damaged central nervous system.

SUMMARY

1. A study of 323 cases of meningococcus meningitis is presented.

2. The mortality in this series was 17.7%.

3. A questionnaire sent to the 268 survivors was returned by 178.

4. Of 22 patients complaining of postmeningitic deafness, 11 reported for a clinical examination during the year 1948.

5. Of the 11 patients critically examined 3 were totally bilaterally deaf; in 2 the hearing was normal in one ear while the other was totally deaf; in 2 the hearing was normal in one ear and deficient in the other; in 2 one ear was totally deaf and the other ear showed markedly impaired hearing; and in the last 2 cases the residual hearing in both ears was markedly deficient.

6. We have discussed the variability of morbidity and mortality and indicated that chemotherapeutic and antibiotic medication may play a role in the decreased mortality of recent years.

7. We have discussed the probable pathology responsible for postmeningitic sequelae.

1340 WELLS BLDG.

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GROVE-FOX

TABLE 1
MORBIDITY AND MORTALITY ACCORDING TO AGE GROUPS

AGE GROUP	TOTAL CASES	TOTAL DEATHS	PERCENT OF DEATHS
Under 1 year	24	3	12.5
1-5 years	69	15	21.7
5-10 years	33	6	18.1
10-15 years	20	2	10.0
15-20 years	21	1	4.8
20-30 years	40	2	5.0
30-40 years	50	8	16.0
40-50 years	30	9	33.3
50-60 years	23	4	17.4
Over 60 years	13	5	38.5
TOTAL: 323		55	AVERAGE: 17.7

TABLE 2
PERCENTAGE OF DEATHS FOR EACH YEAR

YEAR	CASES	DEATHS	PERCENT
1936	8	1	12.5
1937	4	1	25.0
1938	5	5	100.0
1939	4	2	50.0
1940	3	0	0.0
1941	9	2	22.2
1942	8	3	37.5
1943	64	11	17.2
1944	140	20	14.3
1945	78	10	12.8

TABLE 3

RESIDUALS REMAINING FOLLOWING AN ATTACK OF
ACUTE MENINGOCOCCUS MENINGITIS

CLASSIFICATION OF CASES	NO. OF CASES	PERCENT
Total number of cases reviewed	178	—
Residual effects not present	90	50.6
Residual effects present	88	49.4

TABLE 4

TABULATION OF RESIDUAL EFFECTS OF ACUTE
MENINGITIS

RESIDUALS REMAINING	NO. OF CASES	PERCENT
Headache	58	32.6
Muscle pains	37	20.8
Joint pains	30	16.0
Hearing defects	23	12.9
Muscle twitching	21	11.8
Fainting	7	3.9
Convulsions	1	0.56

TABLE 5

TABULATION OF RESIDUALS REMAINING ONE YEAR
OR MORE AFTER THE ONSET OF MENINGITIS

RESIDUALS	NO. OF CASES	PERCENT
Headache	40	22.5
Muscle pains	22	12.3
Hearing defects	22	12.3
Joint pains	15	8.2
Muscle twitchings	9	5.1
Fainting	2	1.1
Convulsions	0	0

TABLE 6

AMOUNT AND DEGREE OF HEARING IMPAIRMENT

Cases Examined	Total Deafness of Both Ears	One Ear Totally Deaf One Ear Normal	One Ear Totally Deaf One Ear Partially Deaf	One Ear Partially Deaf One Ear Normal	Both Ears Partially Deaf
11	3	2	2	2	2

TABLE 7

IMPAIRED HEARING AS A SEQUEL OF MENINGITIS

	NO. OF CASES	PERCENT
Number of patients having on-set of hearing defects with meningitis	14	—
Impaired hearing still present	13	92.8
No hearing defect at present	1	7.2
Improvement with time, but not complete recovery	6	42.8
No improvement with time	7	50.0
Bilateral impairment	8	57.1
Unilateral impairment	6	42.8
Bilateral impairment with difference of degree of impairment on the two sides	5	35.7
Bilateral impairment with no difference of degree of impairment on the two sides	3	21.4
Hearing aid used and of value	2	14.2
Hearing aid not used	12	85.7
Variation of deafness with weather	6	42.8
No variation of deafness with weather	8	57.1
Ability to hear better over telephone	7	50.0
Ability to hear better in noisy room	1	7.2
Ability to hear better when looking at person speaking	6	42.8
Consulted doctor about hearing	11	78.5
Did not consult doctor about hearing	3	21.4

TABLE 8

VARIATIONS IN SYMPTOMS OR AFTER EFFECTS

	NO. OF CASES	PERCENT
Patients with variations in after effects	56	70.8
Patients with no variations	23	29.1
Changes in weather	21	26.6
Working	18	22.7
Exercising	13	16.4
Not indicated	9	11.4
Winter	9	11.4
Resting	5	6.3
After drinking large amounts of water	5	6.3
Summer	4	5.1
After sweating	4	5.1

DISCUSSION

DR. H. MARSHALL TAYLOR (Jacksonville, Fla.): I do not believe we should attribute the deafness entirely to the meningitis itself. There is no question that serum sickness or anaphylaxis in serum sickness can cause deafness. A few cases have been reported as due to meningitis which were due to the anaphylaxis or the horse serum.

DR. CHARLES E. KINNEY (Cleveland): Two years ago, I presented before this Society a series of 29 cases of total deafness following meningitis, not exclusively of meningococcal type. I would like to add 13 cases, making my series now 42.

One of the original 29 patients has now shown some evidence of return of residual hearing. In checking more closely into the history and records on that particular case, I found that the symptoms of encephalitis were more prominent than of true meningitis. This further confirms the observation of Eagleton that the deafness would be less profound and less permanent if encephalitis was the main clinical finding. I have seen three additional cases, since my report two years ago, in which encephalitis was the prominent thing; in all three there is definite recovery of hearing. Also, in one of those three cases the hearing was better at the end of a year than it was at the end of two years, which would further corroborate the statements of Dr. Fox and Dr. Grove.

DR. EDMUND P. FOWLER, SR. (New York): I wish to introduce a thought in regard to milder cases of meningitis. We know that some of the mild cases get total deafness.

There occurs in all kinds of meningitis a great and prolonged sludging of the blood. Sludging of the blood may cause nerve deafness because of the character of the collateral circulation in the labyrinth.

Dr. Henry L. Williams (Rochester, Minn.): You may recall that Hoyne in Chicago reported a large number of cases of meningococcic meningitis from that city. He pointed out that meningococcic meningitis was somewhat of a misnomer. It was primarily a septicemia. He suggested that a good many of the cases of meningitis might be precipitated by the practice of doing diagnostic spinal punctures in doubtful cases.

Might not intrathecal therapy in meningitis be a factor in producing arachnoid granulations and thus add to the sequelae?

Dr. Moses H. Lurie (Boston): I would like to call attention to the fact that two of the audiograms shown indicated middle ear deafness. I don't think that can be blamed on the meningeal infection because that would involve a true nerve deafness.

Dr. W. E. Grove (Milwaukee): In answer to Dr. Lurie, our findings indicated that in our cases we had a mixed form of deafness with a conductive component. How it occurs I am not entirely sure that I can explain. Neither can I state that these patients may not have had a conductive deafness before they had their meningitis. It is certain though that our cases show a mixed form of deafness in some cases in which there was residual hearing.

Dr. Max Fox (Milwaukee): In our series of cases we know that every time we have introduced intrathecally penicillin, streptomycin, etc., we have had a great deal of ill effects. Dr. M. Masto reported cases of encephalopathies, convulsions, etc., as the result of intrathecal therapy. I pretty well agree that intrathecal therapy should be eliminated.

About 28 years ago I began treating meningitis cases for the State of Wisconsin in our fever hospital in the City of Milwaukee. All of our therapy with the Flexner serum was done intravenously and intrathecally and we had a policy of doing all of our work by cisternal lumbar washout. Our death rate at that time was 40-48%. I must say we no doubt invited some distress, but the gradual improvement in recovery rates has made possible the study of these sequelae.

Serum anaphylaxis was one of the most dangerous things we had to deal with. Indeed I am quite certain that serum reactions at that time caused a good many syndromes that were interlocked with transitory deafness but I doubt if they were permanent. In the present series of cases, since the advent of chemotherapy and the antibiotics, we are not using them intrathecally and I do not believe there is anything anaphylactic about it.

In regard to Dr. Kinney's series of 42 cases, I am sure that with the new drugs available, the recovery rate is going to be better.

In supplement to that, I want to mention Bailey's work. The acute fibroblastic infiltration has invited a lot of hydrocephalic cases. It may be that if we have a fibroblastic invasion it requires earlier diagnosis and enthusiastic therapy commensurate with the severity of the disease.

Dr. Peterman and myself have been the authors of a paper on measles encephalitis. A follow-up of 49 cases did not reveal any residual hearing loss. In the last report on a series of 69 cases of bulbar poliomyelitis, I cannot recall a hearing disturbance interlocked with them. We recently reported a series of 27 post-meningococcic meningitis cases, following the Waterhouse-Friderichsen syndrome with a minimal amount of damage in the central nervous system. In this series of cases follow-up letters indicate no personality changes.

Those who are here from St. Louis know what you had to deal with in 1930, 1931 and 1932 in the 14-1500 cases with 22.5% of personality changes in infectious encephalitis with cranial nerve residuals. That is not true of our series of cases. We did not have encephalitic syndromes.

We are certain that in these cases of menigococcic meningitis the fibroblastic infiltration must be interlocked with the eighth nerve difficulty.

EARLY DETECTION OF MIDDLE EAR MALIGNANCY

HOWARD P. HOUSE, M.D.

LOS ANGELES, CALIF.

Carcinoma of the middle ear is rare, as shown by the studies of Newheart,[1] Furstenberg,[2] New,[3] Schall,[4] Spencer,[5] Figi and Hempstead,[6] Robinson,[7] Bowman,[8] Smith,[9] Stokes,[10] Rosenwasser,[11] and others. A survey of the literature reveals 201 authentic cases of this disease have been reported to date.

As early as 1917 Newheart stated that 85% of all cases of malignancy of the middle ear were superimposed on chronically discharging ears. This was later confirmed by Furstenberg in his summary of 75 cases reported in the literature up to 1924. Figi and Hempstead stated but one-third of their cases were preceded by a history of chronic otitis media.

Successful treatment of carcinoma, regardless of its location, depends on an early diagnosis. This is especially true in the middle ear, for by the time the lesion becomes grossly visible, invasion of the underlying bone has often already occurred. The mortality in these cases is then very high, irrespective of treatment.

For this presentation, two basic principles have been assumed. First: the majority of middle ear malignancies are superimposed on chronically discharging ears; second: the successful treatment of middle ear malignancy depends on establishment of a diagnosis at the earliest possible moment.

As pointed out by Diamont[12] it is very difficult to differentiate clinically between granulations in chronic otitis media and early malignancy. Certainly, repeated biopsy of granulations in a chronically discharging ear to continually rule out carcinoma, is not feasible. Likewise, in view of the rarity of carcinoma of the middle ear, prophylactic radical mastoid surgery cannot be performed in all cases of chronic otitis media.

In cytologic studies of vaginal[13] and bronchial secretions[14] the Papanicolaou technique has been of value in determining the presence of cancer before it becomes grossly visible. It is my opinion this same procedure applied to the exudate of a chronically draining ear may provide the otologist with the means of making an early diagnosis in cases of middle ear malignancy. With this in mind and with the cooperation of Dr. H. Russell Fisher, the exfoliative cytologic diagnostic technique was used in the two cases herein reported.

A cotton applicator was used to obtain exudate in the middle ear area and smears were made on standard slides, which were immediately immersed before drying in the standard alcohol-ether fixative. These were stained by a routine Papanicolaou method. The positive smears observed in this study were so identified by the occurrence of clumps of epithelial cells which irregularly exhibited large nuceoli in the enlarged nuclei.
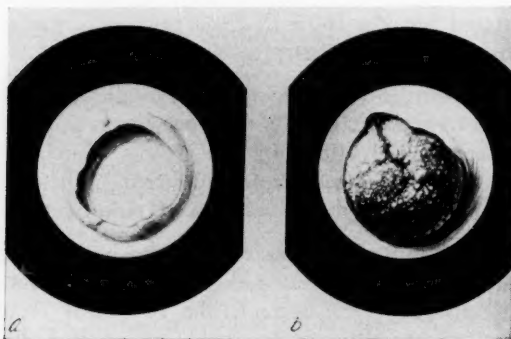
Fig. 1.—a. Destroyed drum and ossicles with dry middle ear, August 1947. b. Large tumor mass visible, June, 1948.

CASE 1.—Mr. R. S., aged 35, was first seen in June, 1945. The history at that time revealed bilateral intermittent draining ears since childhood with exacerbations often occurring in conjunction with upper respiratory infections. There had been no vertigo or ear pain.

The examination revealed both drums were largely destroyed, together with the ossicles. Both middle ear cavities contained rather smooth mucosa interspersed with some small areas of granulation tissue. There was a clear, odorless mucoid moisture present in both middle ear cavities. Audiograms showed a bilateral loss consistent with the pathological changes observed.
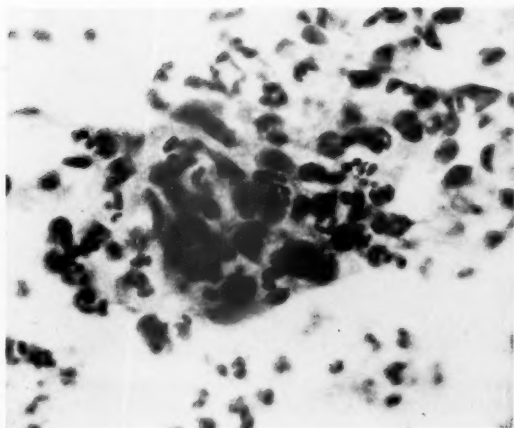
Fig. 2.—Photomicrograph showing postoperative drainage positive for malignant cells within three weeks after surgery.

This patient was observed in June, September, and October of 1945; in February, 1946, and in February and August of 1947. These visits were made for routine observation and often coincided with an increase in ear discharge during the course of an upper respiratory infection. In each instance the middle ear cavities appeared unaltered from previous examinations, and at the time of his last visit in August, 1947, the left ear was completely dry (Fig. 1a).

In June, 1948, eight months following his previous visit, the patient returned complaining of a discharge from the ear of three weeks' duration following a head cold. This was associated with a feeling of fullness and discomfort around the left ear area. Examination revealed an odorless mucoid discharge with a large mass of granulation or tumor tissue nearly filling the entire left middle ear cavity (Fig. 1b). A biopsy was positive for epidermoid carcinoma.

An extensive radical mastoidectomy was immediately performed on July 14, 1948, including removal of the anterior bony ear canal wall, which was invaded by tumor tissue. Frozen sections of the parotid and surrounding tissue indicated the growth had apparently been successfully removed before closure.

During the two months' period following surgery, several Papanicolaou smears were made of the discharge and, surprisingly enough, were positive for malignant cells on August 10, 1948 (Fig. 2). The relatives were informed of the poor prognosis and more radical sur-

Fig. 3 and 4.—Gross autopsy specimen revealing intracranial extension.
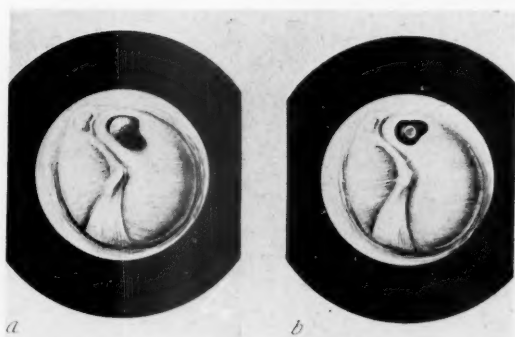
Fig. 5.—a. Small mass resembling polyp presenting from epitympanum.
b. Base of mass visible through perforation following removal.

gery and irradiation were advised, but refused. In spite of these positive smears the postoperative course was uneventful. The ear healed completely, and a dry cavity was obtained some two months following surgery.

In November, some four months after surgery, the patient developed a partial seventh nerve paralysis associated with ear pain. Subsequently, the paralysis subsided but the ear pain persisted. The epithelium over the middle ear area broke down and mucoid drainage was noted with some slight bleeding. Two months later, or six months after surgery, the patient complained of increasing ear pain and a return of the mass was noted in the ear cavity, accompanied by palpable neck glands. On February 23, 1949, some seven months after his radical mastoidectomy, the patient became semicomatose and was admitted to the hospital where he expired two weeks later. Autopsy showed the presence of intracranial extension of the tumor (Fig. 3 and 4).

CASE 2.—Mrs. R. E., aged 34, was first seen in March, 1949, in consultation with another otologist. The patient stated she had had a fullness in her ear two years previously, associated with a slight drainage. Her otologist removed a small polyp at that time from the area of the epitympanum and her subsequent course was uneventful, with complete healing of the small drum perforation.

Two weeks prior to her visit to my office, the patient had again consulted her otologist because of fullness in the ear. His examination revealed a return of what was described as a polyp in the same
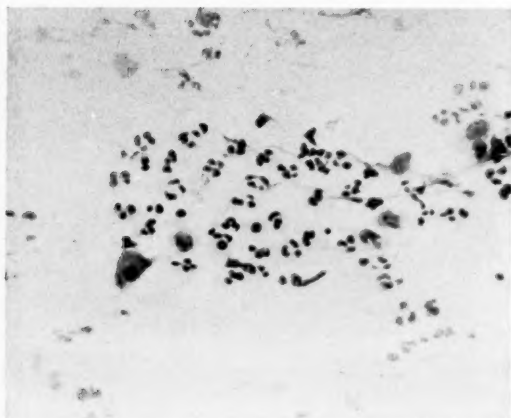
Fig. 6.—Smears from area of epitympanic perforation were negative for malignant cells, as shown in this photomicrograph.

epitympanic area. The surface of the polyp, however, was slightly granular, and bled on manipulation (Fig. 5a). The otologist removed the polyp, which on sectioning revealed an early carcinoma.

My examination on March 1, 1949, revealed a small perforation in the epitympanum with some granulation tissue visible about the base of the recently removed polyp (Fig. 5b). A Papanicolaou stain taken from this area was negative for evidence of malignant cells. (Fig. 6).

On the basis of the previous positive biopsy and in spite of good hearing, a complete and extensive mastoidectomy was performed on March 2, 1949. At that time some granulation tissue was removed from the epitympanum as the only positive finding. Repeated frozen sections during the course of the surgery were negative for malignancy. Likewise, three Papanicolaou smears made at two-week intervals during the postoperative healing period were also negative for evidence of malignant cells. The patient made an uneventful recovery and has a completely healed and dry ear cavity at the present time. It would appear her prognosis is excellent, although she must be observed for a much longer time.

CONCLUSION

The exfoliative cytologic diagnostic technique may have definite value in the early detection of cancer of the middle ear. This tech-

nique may also aid in determining whether the entire lesion was successfully excised at the time of surgery. I realize conclusions cannot be drawn on two such cases, but report them to stimulate interest in this diagnostic and prognostic approach to the distressing problem of middle ear malignancy.

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DISCUSSION

DR. THEODORE E. WALSH (St. Louis): I would like to interject one thought here: In the second case, the first polyp or granulation was, I understand, not sectioned. One wonders whether it had malignant cells in it at that time. My only reason for adding to this discussion is to urge that every piece of tissue that is taken out of a human body be sent to the pathologist. One cannot be sure by looking with the naked eye whether tissue is malignant or benign. I have seen malignancies that were missed because the surgeon was not energetic enough to get the help of his pathologist.

Dr. Joseph A. Sullivan (Toronto, Canada): Dr. House has presented a most timely subject.

I wish to add to his series three proved cases.

I would like to ask Dr. House, before I mention briefly my own cases, how many cases in the literature that he reviewed had in conjunction with the discharge an accompanying facial paralysis? The three cases that I have had, all presented a facial paralysis. One of these patients found her way to the Mayo Clinic and perhaps Dr. Williams will clarify the picture that he found.

I think that the cytological examination of a chronic aural discharge and the pathological examination of every piece of tissue removed are most important. An individual who complains periodically of bleeding from the middle ear is, in my opinion, a case highly suspicious of early malignancy.

Dr. Benjamin H. Shuster (Philadelphia): I have treated several of these patients, two of which illustrate a point which should be brought up.

One was a boy of 16, who had a chronically discharging ear, and another was a man in his thirties who had had a radical mastoidectomy 20 years before he came under my care. In these and the other cases that I have had, the most outstanding symptom was pain. An individual with a chronically discharging ear does not usually have pain. The presence of intense pain suggests the possibility of malignancy.

I do not feel the optimism of most people, that if you make an early diagnosis of malignancy of the middle ear you are going to be able to cure your patient. When the Papanicolaou stain shows the exfoliating carcinomatous cells it is not an early diagnosis any longer. The treatment will largely depend upon the finding of a cure for carcinoma and not particularly in the early stages. The patient in every case that I have seen, whether he had treatment early or late, passed on beyond. While we must be on the lookout to find the early stages we cannot be too optimistic that this is the solution to the problem.

Dr. H. L. Williams (Rochester, Minn.): I have been associated with Dr. Figi in the treatment of certain malignancies of the ear and, as you know, he has had considerable success in treating such malignancies. I believe that the success which he has had can be ascribed wholly to his methods of treatment. If you remember Dr. Aub's talk at the Laryngological Association, you will recall that he pointed out that early diagnosis was not as important as the treatment of the condition.

Dr. Figi has set up as his criteria of treatment in these cases of malignancy of the ear that the bone should be removed widely beyond the area of gross involvement, and that if necessary the dura should be widely uncovered. This often entails the destruction of the facial nerve. The field of operation is carefully seared with diathermy after the removal of tissue and radium needles or packs placed immediately afterward. In other words, as Dr. Aub said, since the only opportunity to cure these patients is the first time they are operated on, Dr. Figi feels that the most heroic measures consistant with survival should be used.

Regarding the patient mentioned by Dr. Sullivan who came to me from him with a letter, to my great horror and surprise at the time of operation for nerve graft I found suspicious looking granulation tissue in the middle ear and upon sending this to the pathologist received a fresh frozen section diagnosis of carcinoma. With the aid of Dr. Figi we went ahead with the operation. It is about two and one-half years now since the procedure, and the patient has no evidence as yet of a recurrence.

One of the most interesting cases of malignancy I had was a patient who one year before coming to us had an acute mastoiditis with fever of 104° F., and a Gradenigo's syndrome. Operation on the mastoid was done several times without any particular relief of the patient's symptoms. She still ran a febrile course with symptoms suggesting duromeningeal irritation. I re-operated on this patient and did an apicectomy. The patient made a fairly good recovery for a while

but then developed a facial palsy. I asked one of our neurosurgeons to do an anastomosis; at the time of operation he cut through some material in back of the ear which was diagnosed on frozen section as a Grade IV malignancy. The pathologist stated that in his opinion the cell was a type which evolved from the mucosa of the middle ear. This appeared to be a case in which malignancy evolved on the basis of an acute otitis media and mastoiditis. In this instance, of course, our success in relief of the patient was not very great. I hope that Dr. Figi will report his cases again. He has a large group and I think they will prove very interesting. I feel that his first report at the Academy opened up a new era in the treatment of malignancy of the ear.

Dr. L. A. Schall (Boston): This paper again emphasizes that anything in the way of a diagnostic method that we can use to make an early diagnosis of malignancy is important.

In 1935 I was rash enough to report on five cases of malignancy of the middle ear in which the patients survived from one to five years. One died at the end of two years, not from carcinoma but from pneumonia. At the end of the next two years they were all dead except one, a case of hemangioma of the middle ear, who had survived since 1930. In the next five years I saw six more cases of carcinoma of the middle ear, and two years ago at the Massachusetts Eye and Ear Infirmary we had six more cases. In the past six months we have had two more. These are distressing cases.

I have laid down the principle of radical mastoidectomy by postauricular incision, with a circular incision of the entire external auditory canal. The auricular incision cannot be made wide enough. I agree wth the essayist in that you cannot determine the extent of the invasion of the mastoid cells by supposition. You must look and see.

I have cases in which I have found cancerous cells and the x-ray interpretations of the mastoids were negative. You must explore and use wide surgery. You must use the full extent of irradiation at the time of operation. Unless you have the bone removed completely, external irradiation does not do any good. You can cause extensive bone necrosis by irradiation and yet the tumor cells will still grow in the remaining bone. So I appreciate the technique suggested by the essayist and I heartily endorse having every specimen examined pathologically, and I especially recommend it when you have a radical mastoidectomy that shows granulations.

Dr. Howard P. House (Los Angeles): I agree with Dr. Walsh that tissue removed from any operated field should be routinely sectioned.

I agree with Dr. Schall that we obviously cannot report the patient in the second case as cured. It will be interesting to note, however, what happens in the future in view of the negative Papanicolaou stains and negative frozen sections at the time of surgery. I cannot accurately answer Dr. Sullivan's question pertaining to facial nerve palsy in cases of middle ear malignancy. It was a late development in three of my five cases.

LXXIV

SURGERY FOR CONGENITAL ATRESIA

WITH REPORT OF A CASE

HARRY C. ROSENBERGER, M.D.

CLEVELAND, OHIO

A year ago before this Society our president, Dr. Marvin Jones, expressed the opinion that a re-examination and reappraisal of otological concepts and tenets might uproot long-held fallacies and thereby promote the advancement of otology. In that spirit I would like to reopen the question of our attitude toward and treatment of congenital atresia of the auditory canal. The condition of acquired atresia will not interest us in this presentation.

Irrespective of whether the reported incidence of congenital atresia is 1 in 1000 or 1 in 2000 aural cases it is likely that each of us has had this problem to deal with in his individual practice. It is likewise probable that each of us has sought help in a review of the literature and thus was inoculated by the beliefs and prejudices he found there.

Historical Background. Although Kiesselbach in 1883 is given credit for the first surgical attack on this anomaly, it was the glowing report of Dean and Gittins[1] in 1917 that stimulated American otologists to attempt aid in behalf of these unfortunate patients. Excepting the restrained optimism of Page it is frankly stated by Dean and Gittins that their surgery was performed in spite of the prevailing pessimism engendered by reports of similar efforts to that time. Fortunately for their patient, for them and for posterity, there resulted a marked improvement in hearing.

Eight years after the optimistic report of Dean and Gittins the subject of congenital atresia of the auditory canal was further elucidated by Beck[2] who emphasized the psychological aspects of the deformity. Beck enlisted the interest of Herman Adler of Chicago and together they appraised the psychological impact on the patient of this congenital defect. Beck also called attention to the

salutary consequence on the parent, of surgical correction of the congenital defect in his child.

In 1931 Fraser,[3] and in 1933 Richards,[4] published informative articles stressing especially the embryologic reasons for the maldevelopment of the three divisions of the ear and particularly the middle and external divisions. About this time Hume and Owens[5] spoke with some enthusiasm of operation in unilateral cases as resulting in appreciable hearing improvement and benefiting the patient psychologically. Up to this time operation was almost universally disapproved in unilateral cases excepting where the deformed ear was the site of an infection necessitating surgical interference. I would point out that this contribution of Hume and Owens was noteworthy not so much for the surgical technique they advocated as for the reversal of prevailing attitude regarding operation in unilateral cases.

In 1934 Hall,[6] and in 1943 Cohen and Fox,[7] reiterate the dictum against operating in unilateral cases of congenital atresia. However, in 1947 a new viewpoint was introduced by Pattee[8] who was impressed by the similarity of audiograms in his cases of clinical otosclerosis and congenital atresia. Because of this resemblance he postulated the hearing defect as related to stapes fixation owing to the deformity and probable fusion and fixation of the malleus and incus. He also was impressed by the suitability of the Lempert endaural fenestration technique for the surgical attack in these cases.

Characteristics of Congenital Atresia. Passing on for the moment to a consideration of the known facts relating to congenital atresia we may reflect on the following. The term "congenital atresia" is a misnomer in that the atresia of the canal is only one of several equally striking features of the anomaly. The terms agenesis or aplasia of the ear would be more fitting and accurate but they lack priority. To quote Fraser, "in a typical case the auricle is malformed or absent, the external meatus is occluded by bone, the malleus is small or absent while the incus is large but misshapen, the tympanum is narrowed in breadth and height and the window niches are occluded by connective tissue . . . [Also] the facial nerve is usually small and may cross the tympanum uncovered by bone." Various combinations and degrees of these deviations may occur together with facial paralysis and facial asymmetry. Fusion of the incus and malleus appears not uncommon. Heredity seems to play little if any part, but curiously the unilateral atresia is much more common among boys than girls, preponderantly favors the right side and occurs much more frequently than bilateral atresia.

Embryologically the cochlear and vestibular mechanisms undergo much earlier development and from totally different anlage than do the middle and external ears and ossicles. Hence the internal ear may be normal but associated with marked agenesis of the middle and external ears and ossicles. Likewise the converse may be true. In this connection Richards stresses the importance of recognition by otologists of the absolute lack of a tympanic membrane in congenital atresia and the consequent futility of a surgical exploration for this structure.

Evidence of Cochlear Function. Before attempting surgical correction of congenital atresia it is necessary, of course, to secure reliable evidence that the cochlea is functioning. In bilateral atresia the patient's ability to speak is indisputable evidence of the presence of some hearing function. However, in unilateral atresia the speech and hearing may appear normal although hearing in the atresic ear is greatly reduced. To ascertain the status of the cochlear function of the involved ear recourse may be had to roentgen examination[9] for mastoid pneumatization and the presence of a tympanum and external auditory canal, tubal inflation for patency, rotational tests for vestibular function and tuning fork and audiometric tests for air and bone conduction. Typically the atresia produces a conduction type of hearing defect unless primary or secondary cochlear damage has occurred.

Reasons for Operating Unilateral Atresia. Let us return now to the original thesis of critical consideration of the dictum that only in bilateral atresic cases with impaired hearing and probable speech defect should be subjected to operation. This idea was basic not only in American but also in European otology for we find the Viennese otologists Alexander, Benesi and Ruttin recommending operation of unilateral cases only if such atresic ears were infected.

In this era of emphasis on hearing conservation it would seem a discordant philosophy that offers help to a child with unilateral conduction deafness of inflammatory origin but denies aid to another child with a similar hearing defect of congenital origin. We rationalize this inconsistency by claiming that one normal hearing ear is quite sufficient and anyhow that little if any hearing improvement is likely to follow surgical treatment of the atresic ear.

While there is an element of truth in the foregoing statement this sentiment is hardly compatible with that of hearing conservation, however large or small the salvaged increment of hearing may be. Even though the postoperative hearing threshold, while improved, remains below the practical level for unaided voice perception the operation may still be considered worth-while. It is bene-

ficial in that the competency of the patient's hearing function is increased the better to withstand the hazards that assail this faculty throughout life. Whereas pre-operatively his economic and social adequacy depended on the function of but one ear, postoperatively he may have two functioning ears even though the operated ear may require a mechanical aid for efficient service.

Another advantage accruing from successful operative intervention in unilateral congenital atresia is that of better appreciation of sound direction. For individuals with good binaural hearing never a thought is given to this faculty. For the person with monaural hearing sound direction perception is an ever present vexation but happily is adjusted to readily and is seldom serious.

Time of Operation. As regards the optimum time at which to operate it would seem desirable in bilateral cases to delay such effort until a reasonable period transpired for the spontaneous development of speech. Postponement until the ear was more developed would also be advantageous since the growth factor would be less likely to compromise the surgical effort. The co-operation of the patient in satisfactorily performing the various functional tests, which of course is desirable, would necessitate an age of at least three to five years and probably nearer the later. In unilateral cases a somewhat later operative date would be permissable as the urgency is not so great. However, too long a delay in operation might conceivably result in diminished cochlear function from lessened use.

Surgical Technique. The type of surgical approach should be dictated by the surgeon's training and judgment. Certainly, however, the object to be attained would favor the employment of the Lempert endaural technique. The surgical objectives may be stated as creating an external canal, preserving an intact tympanum accessible to sound waves through the medium of a pliable skin graft and mobilizing the stapes by removing a fixed, deformed and acoustically useless incus and malleus when present. If Fraser's histological observation that the window niches sometimes may be filled with connective tissue is true, then the added feature of fenestrating the lateral semicircular canal or vestibule theoretically would be indicated as added assurance of endolymphatic movement. Were fenestration contemplated it would be the surgeon's responsibility to provide a suitable covering for the fenestra in the interest both of endolymph protection and mobilization.

REPORT OF A CASE

M. T., a boy aged 2 years, was seen in March, 1946, because of a left acute mastoiditis which required surgical interference. At this

time it was observed that the right auricle was slightly deformed and smaller than the normal left auricle and that the right external canal was absent, being replaced by bone. The parents stated that the child had been slow in acquiring speech but his speech was reasonably understandable for his age.

Because of the dramatic demonstration of temporary deafness during his middle ear and mastoid infection the parents were impressed by the dependence of their son on his one good ear. Consequently they were understandably anxious to increase, if possible, the factor of safety relating to their son's hearing function.

Accordingly tests were done to elicit evidence of cochlear function in the defective right ear. Rotational tests revealed a 25-second nystagmus from each ear and the Weber test was lateralized to the defective ear. The 512 and 1024 d.v. forks, with masking in the good or left ear, were heard by bone conduction in the right or atresic ear in repeated tests. With masking in the good ear the child apparently perceived sound but not speech. Roentgenograms depicted well-pneumatized mastoids bilaterally. Tubal inflation was not attempted.

In May, 1948, the surgical attempt was made to salvage the hearing in the atresic ear. Under general anesthesia the usual three Lempert endaural incisions were made and the mastoid cortex exposed. There was no evidence of an external canal or linea temporalis. The immediate surgical objective was the mastoid antrum. The surgical landmarks used were the posterior border of the glenoid fossa and the adjacent root of the zygomatic process. Allowance being made for the probable width of the tympanum, the mastoid was entered still more posteriorly and the antrum readily located. The lateral semicircular canal was identified with its related crus breve of the incus. The antrum was enlarged just sufficiently to expose the incudomalleolar joint. Palpation showed relative fixation of the incus and its fusion with the malleus and these ossicles were therefore freed and removed en masse with some difficulty (see Fig. 1). The stapes was found to be normally movable and of normal appearance. The bony canal of the facial nerve was not abnormal in size or position. The region of the processus cochleariformis was a smooth mound of bone without a trace of the tensor tendon. The tympanic membrane was lacking and in its position was a solid bony wall. At no time was any structure found which even faintly resembled or indicated either a membrana tympani or external canal.

The mastoid was exenterated but the antrum was enlarged only sufficiently to deliver the malformed ossicles. A split-thickness skin

Fig. 1.—Above: Normal malleus and incus except for absent long process of malleus. Cadaver specimen. Below: Congenitally deformed and fused malleus and incus removed at operation.

graft was cut from the thigh and was draped over a moist cotton stent and fashioned to the mastoid cavity. The three purposes of the skin graft were to provide a substitute tympanic membrane over the antrum thus sealing the tympanum, to ensure a patent external auditory canal and, lastly, to promote rapid epithelization of the mastoid. This technique would be improved by removing only the mastoid structure requisite for access to the tympanum and the creation of an adequate external canal.

Healing was somewhat slow but satisfactory. The antrum is sealed with a pliable membrane and the external canal is spacious. The hearing in the operated atresic ear is quite definitely improved but not to the practical unaided level necessary for normal speech perception. With masking in his good ear the child now readily understands the normally spoken voice at six feet and the whispered voice at one foot in the congenitally defective ear. Certainly his factor of safety as regards his hearing has definitely been increased.

SUMMARY

1. Congenital atresia of the external auditory canal is only one feature of agenesis or aplasia of the total ear.

2. Clinical judgment in dealing with this condition will be facilitated by remembering the separate anlage and variable times of development of the several ear structures.

3. Unilateral congenital atresia occurs more frequently than bilateral, more often among boys than girls and preponderantly affects the right ear.

4. Inferior hearing and faulty speech of bilateral atresia make mandatory a surgical effort at correction.

5. Operation for unilateral atresia heretofore disapproved is indicated and justified on the basis of hearing conservation even though the salvaged increment may require a mechanical aid to render practical service. Sound direction perception in binaural hearing is a minor but appreciated benefit.

6. The optimum time of operation of unilateral atresia is around five years. The preferred surgical technique is the Lempert endaural with removal of deformed malleus and incus if indicated, together with skin grafting to preserve the integrity of the tympanum and the newly created external auditory canal. Fenestration may conceivably be indicated.

10515 CARNEGIE AVENUE.

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DISCUSSION

DR. GEORGE L. PATTEE (Denver): I am very pleased that Dr. Rosenberger has brought up this subject for discussion and wish to compliment him on his paper. I am especially interested in the comments on the unilateral cases and hope that this problem will be discussed further.

Since my paper, in which I reported five ears operated on, (four unilateral and one bilateral) I have done three more unilateral cases. In Denver we also have had one additional bilateral case which was done at the Veterans Hospital by one of the other men in Denver, so that I have now examined and seen ten of these operated ears. All of the patients that I have operated on have had an immediate improvement in hearing in the operated ear but two of them have since lost the gain in function. In one of these, there was an otitis media following the grafting so that she could not be regrafted for some three months. At the time I regrafted the cavity I found that the opening in the antrum was still patent, but the middle ear was filled with scar and granulation tissue. The regrafting was successful but the hearing is the same as before the operation.

I would like to emphasize one point. In the first patient that I operated on I did a large amount of dissection in the mastoid. Later on I felt that it was not necessary to do so much, so I made smaller openings in the mastoid. I had trouble preventing atresia of the canal in the later cases. I am sure if you are going to do the operation, it is well to open the mastoid widely and in addition thin out the edge of the mastoid cortex so as to make a funnel. This allows the scar tissue and graft to attach themselves to bone and prevents some of the atresia that occurs.

In order to demonstrate that the hearing improvement does last I would like to show some audiograms of one of my own patients three years after operation and one of Dr. William H. Wilson's patients operated upon at the Veterans Hospital. My patient, now eight years old, in whom no pre-operative audiograms were obtainable, developed bilateral otitis media one year after surgery. The right ear is still draining and the left ear is healed. It is now more than three years since his operation and these audiograms show that he does have useful hearing.

AIR CONDUCTION HEARING THRESHOLDS POSTOPERATIVELY

	256	512	1024	2048	4096	8192
RIGHT	35	35	40	25	35	35
LEFT	30	30	20	25	3	20

The pre-operative audiograms of the veteran at the Denver Veterans Hospital, show the typical type of hearing in these cases due to fixation of the stapes. This

patient had had an acute mastoiditis in the right ear which was operated upon in New York in 1939.

The left ear was operated on in July of 1948, and the right ear in April, 1949. The audiograms show marked improvement in hearing in both ears. This boy, while still at the hospital after the first operation, could hear the noise of ping-pong balls and running water for the first time.

HEARING THRESHOLDS

		256	512	1024	2048	4096	8192
PRE-OPERATIVE							
A.C.	Right	60	55	60	50	70	75
	Left	40	60	55	40	60	55
B.C.	Right	----	15	0	5	15	----
	Left	----	5	5	0	15	----
POSTOPERATIVE							
A.C.	Right	25	20	40	40	45	70
	Left	35	35	30	30	40	70

DR. FLETCHER WOODWARD (Charlottesville, Va.): I would like to mention a unilateral case that we recently had in which operation was done because the child, aged 4, came in with a subperiosteal abscess. On surgical exploration, using the end-aural technique, we found a well-pneumatized mastoid which was degenerated and filled with pus, atresia of the bony canal, no evidence of membrana tympani, no evidence of the malleus, and the incus deformed. The stapes was normal. The canal was in proper position and the nerve could be visualized in the bone. Our procedure was essentially the same except that we did remove a bit more bone and material.

We tried one thing which may be new. After some ten days or two weeks, we used a new compound now used by the Sonotone Company to take an impression of the ear canal. It is easily prepared, malleable, and can be molded in the cavity. It sets quickly. It was sent off to the Sonotone Company who made a nice plastic mold, which was hollow on the inside and fitted to the concha of the ear. The child can take the thing out himself, wipe it and clean it and put it back in the ear. He will wear that until we have epithelization and healing.

DR. PHILIP E. MELTZER (Boston): We have operated at the Massachusetts Eye and Ear Infirmary about fifteen such patients as described by Dr. Rosenberger. I have collaborated with Dr. Edgar M. Holmes, Chief of the Plastic Service, in some of these cases. It was Dr. Pattee who provided the stimulus for this work. Because of my observation in fenestration work, we changed the technique somewhat. We felt that if the head of the stapes could be exposed rather than merely exposing the antrum, as Dr. Pattee originally advised, the skin graft could be applied directly to the head of the stapes, creating a much better sound-conducting mechanism. Dr. Rosenberger apparently recognized this fact.

Dr. Rosenberger speaks of the endaural approach in this operation. This may be so when there is a reasonably well-developed auricle and evidence of the presence of an external canal. In the majority of these patients there is no such development. All we see is a rudimentary devlopment of the auricle, just a nubbin of cartilage covered with skin with no landmarks whatsoever to indicate the presence of an external canal for the proper outline of endaural incisions. In our experience, one simply makes an incision over the area of the mastoid which will lead directly to the antrum. An important point to be remembered is that the majority of these patients have well pneumatized mastoids. However, occasionally one meets with an acellular, undeveloped mastoid. In such cases a word of warning is extended. If the landmarks are obscured the antrum is found with great difficulty and the structures, particularly the facial nerve, may lie exposed in the depth of the cavity. Unfortunately, we have in our series two patients with facial paralysis. The nerve was never seen because of obscured recognizable landmarks. Fibrous tissue may seal the middle ear space and the nerve may be embedded in this tissue. In an ideal case where there is good cellular development, the cavity should be conical in shape with the base not too wide and the apex directed toward the stapes. This makes an ideally shaped cavity for the skin graft. The apex of this conical shaped graft is in contact with the head of the stapes and is retained with any of the usual materials that will make firm pressure, usually paraffin mesh or paraffin is used for this purpose.

One more comment as regards patients with unilateral, congenital atresia. If the hearing is normal in the good ear, in my opinion the operation should not be done. If one wishes to correct the deformity for cosmetic effect, that is another matter. Later on when the plastic operation has overcome the deformity, one could consider balancing the hearing by performing the operation as described by Dr. Rosenberger.

Dr. HOWARD P. HOUSE (Los Angeles): I would like to throw a little caution into the surgery of these cases because in my experience it is without doubt the most difficult operation I have ever had occasion to attempt. The landmarks are entirely absent with no bony external canal. I agree with Dr. Meltzer in this problem of operating unilateral cases. We recognize that it is an extremely difficult operation to perform and secondly, that certain complications may occur during the surgery. I do not feel that we should operate in these cases if they are unilateral any more than we would operate in a unilateral case of otosclerosis.

I would like to bring up the tremendous value of good x-ray films in certain of these cases. We have Dr. Owens, whom many of you know, who has worked out positions which will show us the presence of the head of the malleus and incus in these cases. This is of tremendous help in making the proper selection. If any of you are interested, I would suggest you communicate with him.

Dr. DOROTHY WOLFF (New York): Through the courtesy of Dr. Lisa and Dr. Risch of New York, we were given a case of congenital malformation of the ear. The appearance of that ear was almost identical with those described by the men who performed this operation.

Our case was of a woman 67 years of age. There was no drum membrane as shown by microscopic examination and no external auditory meatus on the left side. The external ear was normal on the right. There was a malleus and an incus, both of which were malformed. The stapes was quite normal in each ear but there was an anomalous course for the facial nerve in each ear. Furthermore, the facial nerve was extensively exposed without bony covering in these ears.

A second anomaly we have found was a bilateral case in which there was no malformation in the external ear but malformation in the inner ear itself with no proper modiolus, no proper formation of the organ of Corti. An apparently normal malleus and incus with normal drum membrane were present. A stapes with an enlarged head but no footplate was present. Here also an exposed and aberrant course of the facial nerve occurred. The bulk of the fibers joined the

chorda tympani leaving practically none to descend to the stylomastoid foramen. The patient was a seven-month premature infant, female.

In considering surgery the possible aberrancies of the facial nerve must be considered.

Theoretically, a case in which there was a normal inner ear by bone conduction or other tests, even if no footplate of the stapes were present, would certainly be a case in which the Lempert operation could be performed to advantage.

I wonder about the frequency of this anomaly in the male as compared with the female. This woman had successfully covered the defect with long hair. The cosmetic effect is more of a problem with the male, therefore his case is more frequently brought to the physician since it attracts the attention of playmates.

DR. HARRY C. ROSENBERGER (Cleveland): I think our attitude in the past has been characterized by a very large degree of complacency about this condition. There certainly are dangers connected with operating on these patients. It is not easy. One must remember that sometimes the facial nerve has no bony covering whatsoever and there is a risk.

I am not sure, however, that the analogy between this condition and otosclerosis is quite fair. Here we have a child facing life with a residual reservoir of hearing. Yet because the child has good hearing in one ear and we are faced with a difficult operation in order to take advantage of this residual hearing, I think we should think twice before we say, "Well, he can hear with one ear so we just won't bother about the other." I don't believe that this is either fair to the child or a credit to otology.

SURGERY OF MÉNIÈRE'S SYNDROME: EVALUATION OF NEUROSURGICAL AND OTOLOGIC PROCEDURES

FRANK D. LATHROP, M.D. (By Invitation)

BOSTON, MASS.

The surgical management of Ménière's syndrome has received considerable attention from neurosurgeons and otologists during the past decade. As a result, there are now a number of operations which may be employed to alleviate the intractable vertigo and disability of labyrinthine hydrops when medical treatment has failed. All but two are fundamentally alike in that their intent is to disrupt the flow of neurogenic impulses between the vestibular end organs and the vestibular nuclei. Technically, they differ either in their point of attack or the means by which the interruption is accomplished.

The majority of these operations have the labyrinth as their point of attack and effect the desired interruption of the neurogenic impulses by destruction of the vestibular end organs. However, the way in which the destruction is accomplished varies with the procedure. Thus, absolute alcohol may be injected into the perilabyrinthine fluid through the tympanic membrane by way of the oval window as advocated by Wright,[1] or, as employed by Mollison,[2] through a fistula in the horizontal semicircular canal. Cawthorne[3] avulses the membranous semicircular canal through a fistula in the horizontal semicircular canal. An electrocoagulating current may be utilized to effect destruction of the vestibular end organs through a fistula in either the horizontal semicircular canal as practiced by Day,[4] or the superior semicircular canal as advocated by Putnam.[5] Lempert[6] has recently described a technique in which the destruction of the vestibular end organs and the organ of Corti is accomplished by fibrosis as a result of removing the footplate of the stapes and destroying the round window.

Portmann[7] and Passe[8] have devised operations which attempt to stabilize the intralabyrinthine hydrodynamics to effect relief from the intractable vertigo due to labyrinthine hydrops. Portmann's operation, reported in 1927, is said to accomplish this through surgical

attack upon the endolymphatic sac. Passe, in 1948, described an operation in which the stellate ganglion is removed and the vertebral artery ligated to effect the stabilization. To the best of my knowledge, these operations are unique in that they are the only surgical procedures available today which make any attempt to rectify the hydrops of the labyrinth which is now known to be the pathologic condition of Ménière's syndrome.

While otologists have been largely responsible for the foregoing procedures the neurosurgeons have perfected an operation utilizing another point of attack to disrupt the flow of neurogenic impulses between the vestibular end organs and the vestibular nuclei. Dandy[9] employed an intracranial approach to disrupt effectively the nerve pathway between the vestibular end organs and the vestibular nuclei by sectioning the eighth nerve just after its emergence from the internal auditory canal. McKenzie[10] improved the operation by confining the section of the eighth nerve to the vestibular component, thus permitting the pre-operative hearing level to be maintained in the majority of instances. This latter procedure was adopted by Dandy and is now considered by neurosurgeons to be the operation of choice for the surgical treatment of Ménière's syndrome.

The real value of any surgical procedure can be ascertained only after it has been employed in a comparatively large group of representative cases whose postoperative interval is sufficiently long to determine whether or not the desired objective was achieved. Furthermore, it is important when evaluating an operation to obtain a broad over-all picture with respect to the results obtained, technical difficulties and complications encountered, the average duration of hospitalization and the period of economic incapacitation experienced by the patient as a result of the sequelae of the operation. This can be obtained only by careful consideration of the experiences with the operation of surgeons other than the originator. In an effort to evaluate adequately the surgical treatment of Ménière's syndrome the literature was reviewed and additional information obtained from various otologists in Canada, England and this country with respect to their experiences with these operations. In addition, the records of 105 patients who had been subjected to the operations employed for the surgical management of Ménière's syndrome by the Departments of Neurosurgery and Otolaryngology of the Lahey Clinic during the past 13 years were analyzed.

Ideally, the surgical management of labyrinthine hydrops should effectively relieve the patient of vertigo and tinnitus without affecting the hearing. This latter is a debatable point when the disease is thought to be unilateral and the cochlear function of the affected

ear has deteriorated to such an extent that it is no longer serviceable and, as is frequently the case, distorted. In such instances the preservation of a distorted remnant of hearing is undesirable particularly if the hearing in the opposite ear is comparatively good. However, since it has been estimated that approximately 10% of the cases of labyrinthine hydrops exhibit bilateral involvement, Lindsay[11] believes it may be inadvisable to destroy the remaining cochlear function in the more involved ear when the hearing bilaterally is poor unless the symptoms can definitely be ascribed to the ear with the poorer function.

The surgical procedure of choice during the past decade, at least in this country, has been resection of the eighth nerve. When the resection has been confined to the vestibular component of the nerve the results obtained more closely approximate the ideal than any other surgical procedure that may be utilized for the surgical treatment of Ménière's syndrome. Crowe,[12] in 1938, reported a series of 94 patients upon whom Dandy had performed resection of the eighth nerve. The nerve was completely divided in 49 of these patients while the remaining 45 were subjected to differential section or section of the vestibular component alone. The vertigo was cured and the remaining hearing destroyed in those patients in whom the entire auditory nerve was cut. Forty-four of the 45 patients upon whom differential section was performed apparently obtained relief from the vertiginous attacks while the cochlear function was preserved in some degree for, further on in this communication, Crowe stated that 93 patients were cured of vertigo by operation. Apparently the remaining patient still experienced vertigo or could not be traced. The tinnitus was less favorably influenced, for in 25 cases in which the eighth nerve was totally divided it remained unchanged or had become worse in 68%. In his summation, Crowe stated that of 72 cases in which differential section apparently had been employed, the deafness in the affected ear was worse in 22, unchanged in 36 and much improved in 14.

Dandy,[13] discussing the surgical treatment of Ménière's disease in 1941 stated that he had employed either total or differential section of the eighth nerve on 401 occasions with only one fatality. It was his opinion that Ménière's disease could be permanently cured by division of the auditory nerve. Differential section of the eighth nerve was utilized in this series when it was considered necessary to preserve the hearing that remained in the ear to be operated on. It was his experience that the tinnitus disappeared completely in about 50% of the cases and that postoperative dizziness on turning the head may persist for days, weeks or even months. Ireland,[14] in

1948, analyzed the results obtained with the surgical treatment of
Ménière's syndrome as carried out at the Toronto General Hospital
in 168 cases. Resection of the eighth nerve was employed for 90
patients of this series, with one postoperative death. The pre-opera-
tive hearing level in many of these patients was unchanged follow-
ing operation. It was his conclusion that resection of the vestibular
fibers of the eighth nerve is an excellent method of treating those
patients with Ménière's syndrome who fail to respond satisfactorily
to medical therapy when the operation is performed by a well trained
neurosurgeon.

During the last 13 years section of the eighth nerve has been
employed at the Lahey Clinic in 91 patients for the relief of vertigo
due to labyrinthine hydrops. The records of these patients are in-
complete in many instances. However, they contain sufficient in-
formation to permit a decision to be made of the effect of this sur-
gical procedure upon the hearing and vertigo. In addition, a review
of the records allows an estimation to be made of the period of eco-
nomic incapacitation which the patient may be expected to experi-
ence as well as the incidence of postoperative complications and
sequelae for the purpose of comparison with other procedures em-
ployed in the surgical treatment of Ménière's syndrome.

In this series, total section of the auditory nerve was employed
in 25 patients. The vertigo was completely relieved in 15 and re-
mained unchanged in 4. One patient, though relieved of the acute
vertiginous attacks characterizing labyrinthine hydrops, experienced
a constant sensation of unsteadiness after operation which he found
very annoying. Five patients could not be traced. It is difficult to
explain satisfactorily the failure to obtain relief from the vertigo
in 4 of the patients unless the disease was bilateral or the diagnosis
was at fault. The average pre-operative hearing level of 9 patients
on whom audiograms were obtained is graphically depicted in Figure
1. In only 2 of these patients was the hearing of the ear on the oper-
ated side at a serviceable level pre-operatively and all 25 had total
deafness on the homolateral side postoperatively.

In the remaining 66 patients resection of the auditory nerve
was confined to the vestibular component. Forty of these 66 pa-
tients were completely relieved of vertigo, 5 noted no improvement
and 6 complained of being more or less continually unsteady although
experiencing no further acute episodes of vertigo. Thirteen patients
could not be traced. There were 2 postoperative deaths in this
group, one from ependymitis, the other by suicide on the sixth post-
operative day. The latter patient was making an uneventful con-
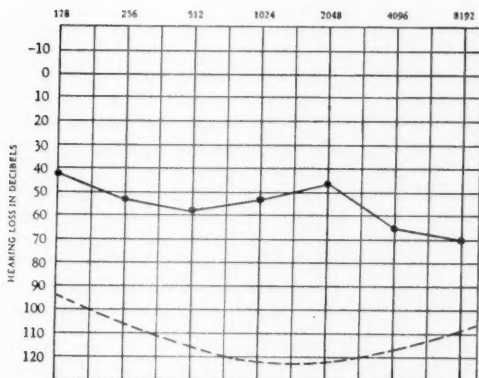valescence following operation and no cause could be determined for

Fig. 1.—Average pre-operative hearing in affected ear of 9 patients subjected to total resection of the eighth nerve.

his action. The fact that 5 of the patients continued to experience attacks of vertigo after operation may possibly be explained on the basis that the vestibular component was insufficiently resected, the disease was bilateral or an error was made in diagnosis. The hearing in 35 of the 66 patients in whom differential section was performed was determined before operation by audiometry. Serviceable hearing was present in the ear on the operated side in only 11 of these patients. Postoperatively, audiometric examinations were made in 10 of these 35 patients and the hearing in the ear of the side on which the operation was performed was found to be improved in 2, unchanged in 4 and worse in 4 when compared with the pre-operative audiograms. Serviceable hearing was present in the affected ear in only 4 of these 10 patients before operation. Following operation a useful level of hearing was found to have been maintained in 2 of the patients while the hearing of a third patient was elevated to this level after operation whereas before operation it had not been serviceable. The average pre-operative audiogram of the 35 patients on whom audiometric examination of the affected ear was made, as well as the average level of hearing before and after operation of the 10 patients in whom similar measurements were obtained, is graphically depicted in Figure 2.

The incidence of postoperative complications in this series has been small. Temporary paresis of the facial nerve occurred in only 2 instances, secondary infection of the wound occurred in 1 patient, and only 1 of the 2 fatalities previously mentioned can be directly
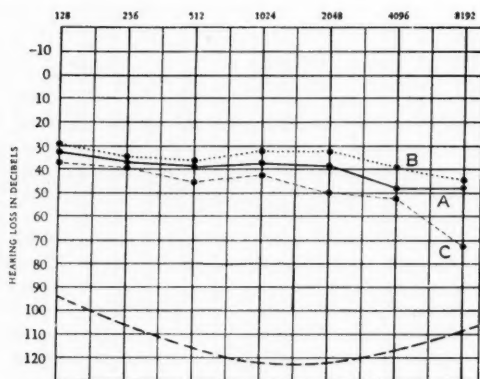
Fig. 2.—Average audiometric level of hearing in affected ear.
A. Before differential section of eighth nerve in 35 patients.
B. Before operation in 10 patients with postoperative audiograms.
C. After operation in 10 patients shown in B.

attributed to the operation. In another patient a cerebrospinal fluid leak developed after operation which promptly ceased following revision of the dural and scalp wounds. The average duration of hospitalization for all patients in this series was 20 days. It must be recognized, however, that a considerable number of these patients were in the hospital several days before proceeding with the operation on the eighth nerve for the purpose of special investigation to rule out the presence of an intracranial neoplasm as the explanation for their symptoms. Furthermore, as experience was gained, the average period of hospitalization deemed necessary gradually decreased to such an extent that during the latter years the average hospital stay was two weeks. It is interesting to note that in no instance was an intracranial neoplasm discovered either as a result of special investigation or at operation. Considerable numbers of references are made in the operative notes with respect to the presence of localized areas of chronic arachnoiditis about the auditory nerve and enlarged vessels pressing on the nerve—so many, in fact, that one wonders whether these findings might not be within the limits of normality. The period of economic incapacitation which patients subjected to this operation experience is apparently of considerable duration. In 12 male patients in whom the operation had been successful the average interval before returning to work was 81 days, the shortest period of convalescence being 27 days and the longest

150 days. While this is admittedly a small number of patients from which to arrive at such a conclusion, it is probably representative of the whole since these 12 male patients constitute 37.5% of the total number of male patients in whom a successful result was obtained. Sequelae of the operation appeared in a moderate number of the patients and were described as weakness, transient periods of postural giddiness and pain, numbness or a feeling of discomfort in the operated area.

Various other operations are available for the surgical treatment of Ménière's syndrome. The surgical procedures of Portmann and Passe endeavor to prevent the acute episodes of vertigo by stabilizing the intralabyrinthine hydrostatic pressure. Although Portmann reported good results the experience of others with this operation has been indifferent and as a result it has never attained any degree of popularity. Passe's operation has been reported too recently to permit any evaluation to be made. However, he reported that 11 of 12 patients were completely relieved of vertigo while the hearing was improved in 10 of the patients.

Destruction of the vestibular end organs by the injection of alcohol into the labyrinth either through the oval window or a fistula in the horizontal semicircular canal has been employed chiefly in England. Wright[15] employed the former method in 60 cases, with improvement in the vertigo in 55 and failure in 5. Total deafness developed in the operated ear in all patients, while the tinnitus was improved in 33, not improved in 16 and not recorded for 9. The remaining 2 patients were free of tinnitus before and after operation. Postoperatively, 2 patients exhibited facial paralyses, one of which was permanent, while meningitis developed in another. Mollison injected alcohol into the labyrinth through a fistula in the horizontal semicircular canal in 50 cases. He reported that 74% of the patients were cured of the acute vertiginous attacks, 10% were not improved, while the remaining 16% were either improved or could not be traced. Mollison employed this procedure only when the hearing in the affected ear was poor and relatively good in the opposite ear. All of the patients experienced further loss of hearing in the operated ear following operation and no postoperative complications were encountered. McDowall[16] employed this procedure in 22 cases. The sudden, severe attacks of vertigo were relieved in 20 patients but transient postural vertigo on sudden change of position persisted in 7. Two patients could not be traced. Tinnitus was absent in 2 patients both before and after operation while it was relieved in 8, improved in 4 and remained unchanged postopera-

tively in the remaining 6 patients of this series. No complications as a result of the operation were encountered.

Lempert's operation for the decompression of the hydrops of the endolymphatic labyrinth in Ménière's disease has just recently appeared in the literature, and, therefore, cannot be adequately evaluated at this time. He claimed that the complete degeneration of the endolymphatic labyrinth and the organ of Corti, which this operation accomplishes by removal of the stapes and the round window membrane, definitely prevents the recurrence of endolymphatic hydrops. He reported that of 10 patients in which this technique was employed, 9 were freed of both vertigo and tinnitus while in the remaining patient the vertigo was abolished and the tinnitus improved.

Avulsion of the membranous horizontal semicircular canal has been perfected by Cawthorne,[17] in England, for the surgical treatment of intractable labyrinthine hydrops. This operation always results in total and irreversible loss of both cochlear and vestibular function of the ear operated upon and is reserved for those cases that are thought to be unilateral and in which the cochlear function is already severely impaired. He reported 159 cases, in 152 of which the cochlear and vestibular function of the operated ear was abolished. Seven patients could not be traced, 140 of the 152 patients were improved and able to work following operation while 12 were not improved and could not work. The only postoperative complication encountered was secondary infection of the wound which occurred in 2 cases. There were no fatalities in this series. Otologists in Canada and this country have utilized this operation in fewer cases and have obtained similar results. Sullivan[18] has employed this procedure for the primary surgical treatment of Ménière's syndrome in 44 cases since 1938 and has obtained relief from vertigo in 43 of the patients. One patient required re-operation, at which time remnants of the saccule and utricle were removed. This patient has since been free of vertigo for six months. He has also avulsed the membranous canal in 4 patients in whom he had previously electrocoagulated the labyrinth because of recurrence of the vertiginous attacks. Following removal of the remaining portion of the membranous canal these patients have likewise been free of vertigo. One postoperative death occurred in this series as a result of a cerebellar abscess. The average period of hospitalization in this series has been seven days. These patients have been sufficiently rehabilitated in three or four weeks following operation to permit nonmanual work, while those patients doing manual labor required four to six weeks for rehabilitation. Cawthorne[17] stated that the 140 patients who

were able to return to their former occupations usually were able to do so in well under two months. Ireland[14], after reviewing 33 of Sullivan's cases for a paper on the diagnosis and treatment of Ménière's symptom complex, concluded that "destruction of the labyrinth is perhaps equally effective [as resection of the vestibular fibers of the eighth nerve] but should be performed by an otologist familiar with minute ear surgery."

Day[19] has employed electrocoagulation of the membranous semi-circular canal and vestibule through a small fistula situated near the ampullated end of the bony horizontal semicircular canal in 55 patients. Fifty-one of the patients were relieved of vertigo while 4 continued to experience dizziness. The persistence of vertigo in 2 of these patients was considered to be due to bilateral involvement of the ears by the hydrops of the labyrinth while in the remaining 2 insufficient electrocoagulation was apparently the explanation since with revision and further electrocoagulation the dizziness was alleviated. The hearing in 35 of the first 40 patients in this series was either totally destroyed or made worse, while in 5 it remained at a useable level. However, in his last 15 cases the hearing has remained at the pre-operative level or became better in 8. In 2 of these, however, vertigo persisted and required a second operation as mentioned previously in order to obtain relief from the vertigo. The previously retained hearing was absent following revision in these patients. Day's experience has been that the tinnitus remains a prominent symptom in those cases in which the hearing has been preserved and was either entirely eliminated or markedly diminished in intensity when the hearing was entirely lost. As a result, there is some question in his mind as to the value of preserving hearing in unilateral cases of labyrinthine hydrops. Postoperative complications of a minor nature occurred in 2 patients in Day's series and there were no fatalities. Two patients developed large hematomas following operation, one of which required evacuation. A great majority of the patients in this series were able to leave the hospital in seven to nine days and were back at work within three weeks.

This procedure has been employed by other otologists with satisfactory results in the majority of cases. Sullivan[18] in Canada has performed 15 such operations with success in 11 and failure in 4 patients. The latter were re-operated and the remaining portion of the membranous horizontal semicircular canal avulsed after the method of Cawthorne with complete relief from vertigo. House[20] operated on 5 patients with this technique with the result that the tinnitus and vertigo were relieved in 2, improved in 2 and made worse in 1 patient. Johnson[21] has performed three such operations
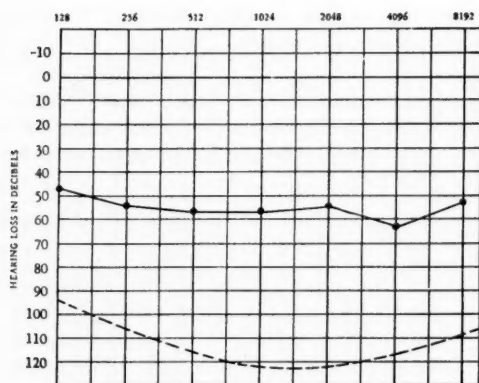
Fig. 3.—Average pre-operative hearing in affected ear of 14 patients subjected to electrocoagulation of the labyrinth.

with complete relief from vertigo while tinnitus persisted and the hearing became worse. Bateman,[22] in England, has utilized Day's operation in 4 cases with freedom from vertigo being obtained in all patients. One patient exhibited a spectacular improvement in hearing following operation. In another, postoperative facial paralysis occurred which disappeared within a month. Furstenberg and Maxwell[23] have employed this procedure in a number of cases with excellent results. The hearing in the operated ear has been lost in each case and all patients have been relieved of the attacks of dizziness. In one patient a facial paralysis occurred as a result of operation with almost complete recovery being obtained in four or five months.

Electrocoagulation of the labyrinth has been employed for the surgical treatment of Ménière's syndrome in 14 patients at the Lahey Clinic during the past 16 months. The vertigo has been completely relieved in all but one of the patients. The character of the dizziness in this patient has been altered and does not, in her opinion, resemble the vertiginous attacks which she experienced before operation. It is described as a "hot flush" having its origin in her stomach. It readily responds to "nerve tonics." The hearing in the operated ear has been totally lost in all patients and in no instance was the hearing in this ear at a serviceable level pre-operatively. The average hearing level for the operated ear before operation is graphically depicted in Figure 3. The effect of the operation upon the tinnitus has been variable. Tinnitus was completely relieved in 3, improved markedly in 5, remained unchanged in 4 and made worse in 1 of

the patients comprising this series. One patient did not experience tinnitus either before or after operation.

There have been no deaths in this series and the only postoperative complication that has occurred is facial paralysis. The incidence of this unfortunate mishap has been relatively high in this small series. Facial palsy was demonstrated in one patient immediately following operation while in another facial paresis appeared on the fifth postoperative day and steadily progressed during the succeeding 72 hours until the face was completely paralyzed. It is interesting to note that at no time during the application of the electrocoagulating current did spasm of the facial musculature occur in either of these patients. Recovery from the palsy was almost complete in both instances four months after operation. However, the patient in whom the facial paralysis was noted immediately following operation presents some mass movement indicating that recovery will never be complete.

The average duration of hospitalization required for this operation for all patients in this series was nine days, the shortest period being six days and the longest thirteen days. There were 7 men in this series, 6 of whom were gainfully employed and 1 was retired. Five of the 6 males who were gainfully employed had returned to their former occupations on an average of eighteen days after discharge from the hospital. The remaining patient returned to work three months from the date of operation. The 7 women in this series were able to perform their ordinary household tasks on an average of fourteen days following their return home. Sequelae of the operation, consisting of transient giddiness on sudden change of position, a sensation of unsteadiness when walking in the dark, or a feeling that the head was foggy, occurred in about half of the cases. These symptoms disappeared within three months following discharge from the hospital.

SUMMARY AND CONCLUSIONS

A number of operations have been developed for alleviating the intractable vertigo of Ménière's syndrome surgically in those patients in whom adequate medical therapy either has failed or the institution of such a regimen would be impractical. The primary purpose of these procedures has been the prevention of the acute episodes of vertigo. The available data tend to indicate that this is most consistently accomplished by resection of the eight nerve and avulsion or electrocoagulation of the membranous horizontal semicircular canal.

Ideally, the surgical management of labyrinthine hydrops should effectively relieve the patient of the vertiginous attacks and tinnitus without affecting the hearing. The results obtained with differential section of the eighth nerve more closely approximate this ideal than with any other method now available. This procedure has been employed in a sufficient number of patients with determination of the hearing before and after operation to permit the conclusion to be made that the pre-operative hearing may be retained in the majority of cases. It is debatable, however, whether the hearing in the affected ear should be preserved when it is poor and that of the opposite ear relatively good. Under such circumstances the hearing is frequently distorted. Retention of such a distorted remnant of hearing is neither desired nor appreciated by the patient and the accomplishment of an operation designed to preserve it which requires a high degree of surgical skill would seem to be an unnecessary surgical exercise. It would seem that differential section would best be reserved only for those patients in whom the hearing bilaterally is good or the disease is thought to be bilateral.

Forty-four of the 91 patients in whom either complete or partial division of the eighth nerve was employed at the Lahey Clinic had audiometric examinations prior to operation. The hearing in the affected ear was of a practical nature in only 13 of these 44 patients. Thus, it may be inferred that practical hearing is not present in the affected ear in the majority of cases in which the hydrops of the labyrinth is unilateral. In such cases employment of an easily executed procedure such as that of Cawthorne or Day is preferable to the technically more difficult operation in which the eighth nerve is divided.

There appears to be little choice between avulsion of the membranous horizontal semicircular canal as practiced by Cawthorne and the method advocated by Day in which the membranous horizontal semicircular canal and vestibule are electrocoagulated. Both are extremely easy to perform as compared to intracranial section of the eighth nerve. They are as equally effective in alleviating the intractable vertigo of labyrinthine hydrops as is resection of the eighth nerve. Cawthorne's procedure may possibly be slightly more difficult of accomplishment than that of Day although I doubt that it is sufficiently so to be given any serious consideration by the experienced temporal bone surgeon. The incidence of postoperative complications appears to be greater in the latter operation when performed by other otologists. The fatality which occurred in one case in which avulsion of the membranous horizontal semicircular canal was performed is of a more serious nature than the temporary facial

palsies that have occurred with electrocoagulation of the membranous canal and vestibule. However, the incidence of postoperative complications in either case has been insufficient to warrant discrimination between them. The available data would indicate that the period of economic incapacitation which the patient experiences is slightly longer after avulsion of the membranous canal than after electrocoagulation.

Since these methods are promising to be as effective in relieving vertigo and the period of hospitalization and rehabilitation is definitely less, it would seem that either of these operations for the surgical treatment of Ménière's syndrome is preferable to the technically more difficult operation of total or partial resection of the eighth nerve when the hydrops of the labyrinth is believed to be unilateral and the hearing in the affected ear is poor.

THE LAHEY CLINIC.

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DISCUSSION

DR. PERCY E. IRELAND (Toronto, Canada): In my paper which was quoted I said that I thought perhaps the best relief for hydrops of the labyrinth was resection of the eighth nerve because of the fact that there was more chance of preservation of hearing. I still have some doubts. I think perhaps that Ménière's, if not in all cases, is a progressive disease.

I have seen one patient who was operated on in 1935 by a partial resection of the eighth nerve. At that time he showed marked improvement in hearing. I saw him last year. He was entirely free from dizziness until two years ago when he developed dizziness again. On re-examination he was completely deaf in the ear that had been operated upon. We felt his dizziness was due to a Ménière's from the other side.

The immediate results are good after nerve section but we are in the process now of bringing these cases back to check final results and we are finding the results of partial resection of the eighth nerve very disappointing. I think we will probably veer to the other operation.

DR. STACY R. GUILD (Baltimore): My experience with Ménière's disease has been that of an observer, from the laboratory standpoint, of Dr. Walter Dandy's long series of 692 cases. There was only one postoperative death, and that was not due to the operation. In the Otological Laboratory, the hearing of almost all of Dandy's Ménière patients was examined both pre- and postoperatively. The hearing level, pre-operatively, has not been studied statistically, but my impression is that the average of the audiograms would be much like that shown by Dr. Lathrop. I personally regard it as desirable to preserve that much hearing, and I know Dr. Dandy likewise thought it important to preserve the hearing of the operated ear, because of the possibility that the other ear may later become deaf, either from Ménière's disease or from something else. Although the loss may be 35, 40, 45 db or more, a hearing aid can still provide usable hearing for such a person. About 5% of the patients who had operations for typical unilateral Ménière's syndrome have returned later with involvement of the other ear by a similar condition.

The final evaluation of methods of treating patients who have Ménière's disease will, of course, have to be on the basis of the long time follow-up studies, such as Dr. Lathrop has presented. In this connection, Dr. Lathrop spoke of Dr.

Crowe's paper of 1938. That report was not based on questionnaires, but the patients were all brought back to the hospital, and were re-examined by Dr. Dandy and re-examined otologically. At the present time another follow-up study of the patients operated on by Dr. Dandy for Ménière's disease is being made, by Dr. Douglass of the Otolaryngological staff and Dr. Green of the Neurosurgical staff. This new study will be of much interest in connection with the topic Dr. Lathrop has so ably discussed today.

DR. HENRY M. GOODYEAR (Cincinnati): I believe that nerve resection should be saved for only those cases in which we have rather good hearing. I do not believe that the evulsion of the endolymphatic membrane or the use of electrocoagulation is necessary in these cases nor does it represent the simplest method of dealing with this syndrome.

Some eight years ago I reported a procedure which I have used since that time in this operation without either evulsion or electrocoagulation and which is simple and satisfactory. I do not believe it is possible to destroy the utricle without destroying the saccule and complete function of the cochlea.

In this operation I follow a posterior incision with removal of enough cortex to expose the semicircular canal well forward. The opening is made in a location similar to that for fenestration and by means of a small dental finder or pick, the utricle is ruptured and the pick turned toward the ampulla of the superior and horizontal semicircular canals. Nothing further is done. My patients have all lost their hearing completely within a week after the operation. However, they have had no recurrence in the operated ear. I feel that in the hands of some, electrocoagulation has added some danger to the facial nerve without any particular advantage.

DR. KENNETH M. DAY (Pittsburgh): There are a number of points brought up by Dr. Lathrop's excellent paper which I think should be commented upon. The first one is the number of operated patients still having dizziness.

The first thing we have to bring out is what are they operated for? We are still too loose in defining Ménière's syndrome. The operation is for hydrops of the labyrinth. We can absolutely differentiate hydrops of the labyrinth from other conditions by the symptomatology; if they don't have, along with the hearing loss, a distortion of hearing, paracusis, and hypersensitivity to loud sounds, which is really a marked recruitment, then they don't have a true hydrops.

I wonder whether some of the failures to cure the vertigo are not due to the fact that the operations were done for some type of vascular vertigo.

Now as to the question of cures, with all due respect to Dr. Dandy and the previous speakers, eighth nerve section does not cure hydrops of the labyrinth. The hydrops of the labyrinth is still there, and is evidenced by the roaring tinnitus which continues afterwards. I have been operating for ten years, varying my technique, always with the ultimate idea of trying to save the hearing and I have saved the hearing in a number of cases. I am not too happy about having saved the hearing if the hydrops is still present. They still have a distortion, a paracusis, and are unhappy and say, "Doctor, I wish you would get that roaring out of my ear."

When I relieve the roar it will not stop the high-pitched tinnitus, but I have yet to have a patient complain of the high-pitched tinnitus.

I have now used the electrocoagulating needle in some 65-70 cases, and I have yet to have difficulty with it. If you use a fine needle and know where you are putting the needle, I see no reason why you should endanger the facial nerve. You must use the proper amount of current. I use a cooking current but not to such a point that all the fluid will boil away and the facial nerve be damaged.

I think the convalescence is the shortest of any present accepted form of treatment. My patients are usually back in gainful occupations within three weeks.

Dr. Samuel Rosen (New York): I wish to mention a new approach to this problem. In the fish the sensations of both equilibrium and sound are carried by the seventh and ninth nerves. They do not have a cochlea or an eighth nerve. The chorda tympani nerve and Jacobson's nerve are the derivatives of this primitive sonic and equilibratory system in the lower animals. The nervus intermedius, which runs between the facial and the vestibular nerves in the internal auditory canal, and is a continuation of the chorda tympani nerve, sends a communicating branch to the vestibular nerve according to Cunningham's Anatomy. In Costen's syndrome where there is malfunction of the temporomandibular joint, patients have tinnitus, vertigo, deafness and occasionally pain in the tongue on the homolateral side. This is thought to be due in part to pressure of the jaw joint on the chorda tympani nerve.

With this concept in mind and knowing that the tinnitus disappears in many of the successfully fenestrated otosclerosis cases, also occasionally after the so-called tympanosympathectomy operation, I attempted a new procedure in the following case: A female 25 years old presented herself 14 months ago. During the past year and one-half she had unpredictable, sudden violent attacks of vertigo, nausea, vomiting and severe roaring tinnitus. I combined fenestration and denervation of the tympanic plexus together with section of the chorda tympani nerve. This preserved the labyrinth. Since the operation 14 months ago there has been a total absence of tinnitus and vertigo. If this procedure had failed the membranous labyrinth could have subsequently been destroyed by electrocoagulation.

Dr. Frank D. Lathrop (Boston): I will not attempt to answer each individual discusser's subject separately. The questions as to whether Ménière's syndrome is a progressive disease and whether we should attempt to preserve hearing because it is a progressive disease since the hearing will ultimately be lost, seems to me to beg the point. It would be extremely difficult for me to advise an individual who has serviceable hearing in the conversational range to have the labyrinth destroyed, with total loss of hearing, when I know that that hearing can be preserved for an indefinite period of time by partial section of the nerve. No one knows how long that hearing is going to be maintained.

By analogy, I would not hesitate to advise fenestration for its beneficial results simply because progression of the otosclerosis would cause further loss of the hearing.

Dr. Day's point in respect to errors of diagnosis is extremely important. We should be very careful in making the diagnosis.

With respect to the postoperative complications, unfortunately, any operation is evaluated by the experiences of a number of individuals with that operation. In Dr. Day's hands facial paralysis has not resulted; in other hands there have been cases of facial paralysis. Unfortunately, there are those who will perform the operation whether they are qualified to do so or not, with the result that the number of complications is likely to increase. As far as I can determine, the only advantage that Cawthorne's operation has is that it is practically impossible for a postoperative complication to occur through any error in technique.

LXXVI

IRRADIATION OF THE NASOPHARYNX AND THE PREVENTION OF DEAFNESS

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BALTIMORE, MD.

Some six years ago a clinic for the prevention of deafness in children was begun in Hagerstown, Maryland. This clinic has functioned as an experimental station for study of the problems involved in a public health approach to the prevention of deafness. The present paper marks the sixth in a series of analyses of various phases of this work.[1-5]

The program for the prevention of deafness carries out the following functions:

1. To discover those suffering from hearing impairment.

2. To discover those suffering from ear or respiratory disorders predisposing to hearing impairment.

3. To provide consultation facilities for any obscure ear or respiratory problem.

4. To give radium treatments to the nasopharynx when they are needed and not otherwise available.

5. To advise, and arrange for, other indicated investigations or treatments.

6. To follow patients over long periods of time to evaluate various forms of therapy, and to improve existent methods.

7. To provide hearing aids and arrange for rehabilitation of those not amenable to therapy.

Each year every child in the third and sixth grade is given an individual screening hearing test. All children found to have a hearing impairment are given appointments for the clinic. At the same time all children who suffer from recurrent or chronic ear or respiratory infections, or who have speech defects, or who are unexplained behavior problems are also given appointments for the clinic. In

SOURCE OF REFERRALS OF CLINIC PATIENTS
(HAGERSTOWN)

	No.	%
Screening	269	26.8
Nurse	180	17.9
Teacher	77	7.7
Private physician	423	42.1
Others	55	5.5
	1,004	100.0

Table 1.

addition to this, referrals for any of these causes are encouraged from private physicians, public health nurses, and teachers.

During the first four years at Hagerstown almost half of the referrals were from private physicians; a little over one quarter were referred from the screening program; the remainder were referred from other sources (Table 1).

All patients coming to the clinic, except very young children, are given full hearing tests, and each receives a complete examination of the ears, nose and throat, including electric nasopharyngoscopic examination.

A report of the findings, along with the clinician's conclusions and recommendations, is sent to the private physician who ordinarily attends the patient or who referred the patient to the clinic.

If radium therapy is indicated and is not otherwise available, these treatments are given at the clinic. Other therapy can be arranged by the private physician or by the public health nurse. If financial help is needed or if the parents fail to realize the desirability of the recommended treatment the public health nurse endeavors to solve these problems.

All patients are followed for as long as possible. During the first four years at Hagerstown the average follow-up for 687 patients was 16.4 months. This includes a great many patients still being followed. Three hundred and sixty-two other patients were seen only once either because no significant ear or respiratory disorder was discovered or because of failure to obtain the patient's co-oper-

HAGERSTOWN CLINIC

FOLLOW-UP	% OF TOTAL	DISPOSITION	% OF TOTAL
No follow-up	34	Maximum benefit	12
0- 6 months	18	No co-operation	38
7-12 months	14	Still under supervision	35
13-24 months	16	Other	15
2- 4 years	17		
4 years +	1		

Table 2.

ation. One hundred and eighty-one patients had been followed for more than two years and 59 for more than three years. More than a third of the first thousand patients seen at Hagerstown was still being followed at the end of the first four years (Table 2). Figures on follow-up for the past two years are not presently available.

This follow-up is an essential part of the program. We know from long and painful experience how frequently relief from ear or respiratory diseases is only temporary. Unless regular repeat examinations are carried out, not only may ground once gained be lost, but a patient may suffer irreversible damage before voluntarily seeking re-examination. In addition to this the only method of evaluating our therapeutic efforts and effecting improvements in the program is through systematic regular studies of long term results.

The use of irradiation therapy for infected adenoids originated in Dr. Crowe's clinic at The Johns Hopkins Hospital in 1924.[6] During the quarter of a century since that time, thousands of patients have been so treated and large numbers have been followed for many years.

No matter how adequately adenoids are removed surgically, lymphoid tissue is a normal constituent of the nasopharyngeal mucous membrane and will recur and hypertrophy under the stimulus of infection or allergy. A small amount of lymphoid tissue in Rosenmüller's fossae, if infected or enlarged, can be responsible for hearing impairment or recurrent ear or respiratory infections.

The normal defenses of the respiratory tract, the continuous sheet of mucus and the cilia which move it at a rapid rate continuously towards the esophagus provide a remarkably impregnable for-

IMPAIRMENT THOUGHT TO BE ATTRIBUTABLE TO	NO. PATIENTS	% OF TOTAL
Upper respiratory and ear infections	551	83
Unknown	64	10
Congenital	15	2
Nerve degeneration	14	2
Stenosis external ear	7	1
Meningitis, etc.	6	0.9
Otosclerosis	4	0.6
Trauma	2	0.5

Table 3.

tress against the invasion of infection. The weak link in the chain consists of the crypts and crevices of the lymphoid tissue where pathogenic bacteria or the common cold virus may lodge and start its invasion of the organism.

Lymphoid tissue is fortunately so sensitive to irradiation that small doses suffice to reduce the size of nodules, flatten their crypts and crevices and discourage subsequent hypertrophy.

Anyone who has used irradiation of the nasopharynx for two or three years becomes so familiar with its beneficial results that he may be tempted to rely too much upon it as a panacea. Its use should be confined to those whose symptoms are clearly traceable to the presence of infected adenoid tissue. These patients fall into clear-cut categories:

1. Those with hearing impairment due to lymphoid tissue obstructing the tubal orifices.

2. Those with recurring acute respiratory or ear infections originating in infected adenoid tissue.

3. Some patients with bronchial asthma on a basis of bacterial allergy.

Routine use of irradiation following tonsillectomy is neither desirable nor justifiable.

Irradiation of the nasopharynx in the presence of chronic sinusitis, chronic otitis media or chronic tonsillitis without proper care of the chronic disease is doomed to failure in most instances.

APPROXIMATE LEVEL IN BEST EAR, PURE TONE AUDIOMETRY	% OF TOTAL PATIENTS	
	FIRST AUDIO.	WHEN LAST SEEN
0-20 decibel loss	28.6	63.0
25-30	48.9	20.3
35-50	17.1	6.5
55 or more	3.4	3.5
Total loss	0.7	0.7
Loss only on 8192 or above	1.3	6.0

Table 4.

The principle of prevention of deafness rests upon the hypothesis that in most instances hearing impairment follows upon respiratory or ear disease amenable to therapy. One of the most important facts to come from study of the Hagerstown figures is that in 4 of every 5 children with impaired hearing the difficulty is traceable to respiratory or ear infections. If we have reduced the frequency or severity of the common acute respiratory infection and its complications we have taken the biggest possible step in the prevention of deafness.

Analysis of 663 patients with hearing impairment showed that in 83% the impairment was thought to be attributable to ear or respiratory infections. This means that in only 1 of every 5 is the deafness not preventable (Table 3).

Our figures also show that in many patients, even when impairment has occurred, it is reversible. Whereas only 28.6% of the patients seen at the clinic had normal hearing in the best ear when first seen, 63.0% had normal hearing in the best ear when last seen. Those who returned to normal had mild to moderately severe impairment when first seen. The patients whose impairment was very severe when first seen had chronic irreversible changes and improved little if at all after therapy (Table 4).

It is difficult to analyze changes in hearing considering both ears. A rough analysis of this sort is shown in Table 5. Those in Group A had perfectly normal hearing in both ears and those in Group I were totally deaf in both ears. The intervening letters signify various grades of impairment in the two ears. All those figures above the diagonal improved. Those in the diagonal remained the same, and those below grew worse.

INITIAL AUDIOGRAM

	A	B	C	D	E	F	G	H	I	TOTALS FINAL AUDIOGRAM
A	29	36	17	16	26	3	0	0	0	127
B	1	21	4	23	28	6	0	0	0	83
C	1	0	2	12	12	2	1	0	0	30
D	0	1	1	26	23	3	1	0	0	55
E	0	0	0	2	33	4	1	0	0	40
F	0	0	0	0	3	11	0	0	0	14
G	0	0	0	0	0	2	4	4	0	10
H	0	0	0	0	0	0	0	1	0	1
I	0	0	0	0	0	0	0	0	1	1
	31	58	24	79	125	31	7	5	1	361 GRAND TOTAL

FINAL AUDIOGRAM (left vertical label) · TOTALS INITIAL AUDIOGRAM (bottom label)

Table 5.

Patients suffering from ear or respiratory infections underwent an improvement following radium therapy comparable to the improvement in hearing.

It is interesting to note that in 268 patients who received no therapy at all, 30% of the ears showed normal hearing when first seen and 54% showed normal hearing when last seen. These represent patients in whom acute or transient ear or respiratory difficulties were responsible for the impairment. Of 365 patients who failed to improve without therapy and received irradiation of the nasopharynx, only 17% had normal hearing when first seen and 50% had normal hearing when last seen. This group represents those whose difficulties were not on an acute or transient basis. Those who had tonsillectomies and adenoidectomies showed the poorest tendency to return to normal hearing, only 43% of this group falling in this category when last seen. Of 1548 ears, or 774 patients, 23% had normal hearing to begin with and 49% normal when last seen (Table 6).

A recent analysis of 459 of my own private patients treated with radium during eight years of private practice in Baltimore shows results comparable to those found in Hagerstown. The age distribution in this group is different, there being a larger percentage of

HAGERSTOWN CLINIC
HEARING CHANGES IN RELATION TO THERAPY

	Total 1548*		No Radium 818		Radium 730		c̄ T and A 458		s̄ T and A 1090		No R̄ 536	
	initial	final	initial	final	initial	final	initial	final	initial	final	initial	final
Normal Hearing	23*	49	29	48	17	50	21	43	24	51	30	54
High Tone Loss	9	15	9	19	10	12	9	22	10	13	9	16
Slight Impairment	23	15.3	26	20	20	13	24	16	23	16	28	19
Moderate Impairment	25.6	10	20	5	31	14	28	12	24	10	18	3
Severe Impairment	15	7	12	4	18	9	15	6	15	7	11	4
Very Severe Impairment	3.4	2.7	3	3	4	2	2	1	4	3	4	4
Total Loss	1	1	1	1	1	1	1	1	1	1	5	5

* Number of Ears   *% of Group

Table 6.

adults, and the follow-ups on the average are not so long. One of the great advantages of a small city public health clinic is the facility with which long follow-ups may be obtained (Table 7).

Many of these patients have been treated only recently but all have been included for the sake of completeness. Fifty-seven per cent of those treated were completely relieved of their chief complaint or markedly improved. This represents 72% of those with adequate follow-up. In many of these patients other forms of therapy were also used. Radium is an adjunct to the standard forms of otolaryngologic treatment (Table 8).

The determination of success in the prevention of deafness and chronic ear and respiratory infections is a more difficult and perhaps impossible task. Comparison of the prevalence of such difficulties in the new patients coming to the Hagerstown clinic from year to year should be an index of the new cases of such disorders developing each year in the county. Changes that occur may or may not be attributable to the activities of the clinic, the advent of chemotherapy, or other more obscure causes. One factor to which such changes may, in some way, be related is the difference in the ages of patients coming to the clinic from year to year (Table 9).

During the first year of the clinic 12.1% of the children screened in the third and sixth grades were found to have a hearing impairment. Four years later only 2.3% of the children in these grades were found to have hearing impairment (Table 10). Similar surveys elsewhere have not shown a comparable decrease in the prevalence of hearing impairment.

DONALD F. PROCTOR

459 PRIVATE PATIENTS

AGE	NUMBER	FOLLOW-UP	%
		No follow-up	26.5
0-2	36	1 month	8.6
3-5	56	3 months	12.4
6-12	95	9 months	10.2
13-18	22	2 years	20.8
19-30	96	3 years	6.3
31-50	133	4 years	6.8
51—→	21	5 years	6.5
		6 years	1.9

Table 7.

During the first year 200 new patients were seen with moderate to severe hearing impairment. During the second year this number had fallen to 158, in the third year to 94 and in the fifth year to 61, less than a third the number found in the first year.

In the same period of time study of the new patients showed a corresponding fall in the numbers of patients with chronic ear or sinus infections from 35 to 11 and from 67 to 29 respectively.

In addition to these drops in the actual numbers one sees a decline in the percentage of those patients who were found to be suffering from the more severe chronic manifestations of ear or respiratory disease. Thus in the first year 25.1% of the new patients had normal hearing, in the fifth year, 50%. In the first year 47.3% had moderate to severe hearing impairment, in the fifth year, only 26.5%. In the first year 39.6% had normal ears, in the fifth year, 57.5%. In the first year 6.6% had evidence of chronic ear infection, in the fifth year, 4.3% (Table 11).

During the first year well over 30 new patients were seen each month. During this past year only 20 new patients have been seen each month.

During the past year irradiation of the nasopharynx has been said by some to be dangerous to the patient. This criticism has been based on two statements; one, that the tissue dosage delivered is

459 PRIVATE PATIENTS
COURSE

% OF TOTAL PATIENTS	CHIEF COMPLAINT	CONDITION WHEN LAST SEEN	% OF GROUP
		No follow-up	21.5
	Upper	Well	26.4
70	Respiratory	Improvement	34.9
	Infection	Questionable	6.6
		No change	10.6
		No follow-up	25.7
	Hearing	Well	18.9
23.5	Impairment	Improvement	26.6
		Questionable	2.0
		No change	26.8
		No follow-up	8.9
	Other	Well	31.7
20.8	Ear	Improvement	22.1
	Complaints	Questionable	8.4
		No change	18.9
		No follow-up	21.7
		Well	26.4
	Total	Improvement	30.7
		Questionable	6.0
		No change	15.2

Table 8.

dangerously large, and the other that even if no immediate damage is done by this dosage there may be a carcinogenetic effect manifested many years later.

It is difficult to calculate the tissue dosage delivered by a radium applicator of this sort. Through the combined efforts of several experts in the field of radiology, chiefly Dr. Bernard T. Feld of The Massachusetts Institute of Technology and Dr. Russell Morgan of The Johns Hopkins Medical School, it has been calculated that the maximum dosage in 10 mg hours with 0.3-mm Monel metal filter is approximately 1600 r. It is probable that the dose is much less than this because of air spaces between the applicator and the tissue treated. This dosage to such a small area is thought to be well within safe limits. Besides this theoretical opinion we have two other evidences that the dosage used is safe. First, the dose was chosen by the late Dr. Curtis Burnam[7] who determined the erythema dose for

AGE OF NEW PATIENTS SEEN IN HAGERSTOWN
% OF TOTAL

Year	Pre-school	1 & 2 grade	3 grade	4 & 5 grade	6 grade	7 grade to 20 years	21 or older
1943-44	24.2	14.0	10.5	15.4	8.8	17.9	9.1
1944-45	14.6	18.3	12.8	16.4	12.2	13.5	12.2
1945-46	12.3	9.9	23.4	18.5	19.7	9.9	6.3
1947-48	21.6	22.3	21.6	11.5	8.6	11.5	2.9

Table 9.

mucous membrane. We have never used a dose as high as this erythema dose. It should be borne in mind that the erythema dose for mucous membrane is much higher than that for skin. Second, careful observations over a period of 25 years have failed to show a single instance of a patient who has suffered any serious damage attributable to the treatment.

As to the possibility of a carcinogenetic effect, I can only say that many thousands of patients have been treated and no evidence of such an effect has yet been found in our patients.

Most of the patients have not been followed more than 6 years but some have been followed for 15 years or more. It was the opinion of Burnam and it is the opinion of the majority of radiologists whom I have consulted that there is no reason to fear a carcinogenetic effect from this form of therapy. Time alone can tell definitely if these opinions are correct.

Although there is every reason to believe that this form of therapy is perfectly safe and there is no well substantiated reason for believing it to be dangerous, this should not lead to carelessness. Increase in the present dose may be dangerous. More than two courses of treatment may be dangerous and certainly more than three courses of treatment would be dangerous. All patients so treated should be followed for many years in order to confirm or refute our claim that no deleterious effects are occurring.

I would like to say in closing that medicine has arrived at the time when an ever increasing effort must be directed towards prevention of disease. The Maryland State Program (now involving four counties) is primarily aimed in this direction. In order for this program to succeed two things are essential. The public health services must work in the closest co-operation with the private physicians of the community and with the school system; and all of us

SCREENING (HAGERSTOWN)

YEAR	NO. SCREENED		HEARING LOSS			
	3rd Grade	6th Grade	3rd Grade		6th Grade	
			No.	%	No.	%
1943-44	372	410	48	12.9	47	11.5
1944-45	962	1014	60	6.5	77	6.9
1945-46	1081	1126	43	4.0	51	4.4
1946-47	971	896	25	2.6	27	3.0
1947-48	1077	1074	20	1.9	30	2.8

Table 10.

FINDINGS IN NEW PATIENTS SEEN IN HAGERSTOWN
IN INDICATED YEARS

Number and % of total seen in year

Year	Moderate to Severe Hearing Impairment		Chronic Tonsillitis		Chronic Sinusitis		Chronic Ear Infections	
1943-44	200	47.3*	126	64.0*	67	23.5*	35	6.6*
1944-45	158	58.5	78	66.0	30	18.4	20	6.5
1945-46	94	61.0	36	60.0	12	14.8	13	8.9
1947-48	61	26.5	46	35.7	29	20.8	11	4.3

* %

Table 11.

together must be searching diligently for early reversible ear and respiratory disorders in order that these may be alleviated before the chronic irreversible difficulties, so commonly seen in adult life, have developed.

THE JOHNS HOPKINS HOSPITAL.

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MEDICAL CARE AND PROPHYLAXIS IN HEARING LOSSES
WITH SPECIAL ATTENTION TO ALLERGIES

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Prior to the advent of the sulfonamides, penicillin, streptomycin and other antibiotics, otology was considered primarily a surgical specialty and treatment aimed at the prevention of deafness was almost entirely surgical.

Since these therapeutic agents have come into general use, however, the character of the entire practice of otology has changed to the point where surgery now plays only a secondary role in preventing hearing loss.

The removal of tonsils and adenoids, submucous resection of the nasal septum, surgical treatment of chronic infective sinusitis and early paracentesis for acute middle ear abscess, are still considered important as prophylactic measures in potential deafness. Mastoid surgery for acute otic infection of the temporal bone, however, is pretty much a thing of the past.

Our change in otologic thinking has been greatly influenced by evidence which has accumulated over the past several years pointing to unbalanced diet; vitamin, mineral and protein deficiencies; glandular dysfunction; psychology factors; inherited predisposition; and allergy as playing vital roles in the etiology of various types of deafness.

The fact that otology was considered predominantly a surgical specialty for so many years may be one reason why the medical approach to the prevention of deafness has been so long neglected and why allergy as a possible etiologic factor has received so little attention.

Since time does not allow for detailed consideration of all of the medical aspects of the subject, this discussion will be limited to the allergic phase of the problem.

Prior to the important investigations of Dohlman[1] in 1942 and Koch[2] in 1947, mention of allergy as an etiologic factor in deafness was limited to the reporting of isolated cases. No scientific data had been presented establishing the fact that allergy could affect the eustachian tubes and the middle ear.

In 1929, Lewis[3] reported a group of six patients with acute otitis media all of whom presented symptoms which strongly suggested to him an allergic type of reaction. The ear lesions were characterized by earache, red and bulging tympanum and discharge of serous fluid. There was no elevation of temperature and no leukocytosis. No examinations for eosinophils were made, either of the discharge from the ear or from the nose.

In 1931, Proetz[4] reported a series of ten similar cases, but in none of his cases did spontaneous rupture of the drum occur. Most of his patients were under five years of age, the ones with the most pronounced symptoms being recently weaned infants whose sensitivities were traced to some component of their new diet. He states that allergic ear manifestations may be recognized by the nature of the onset often accompanied by allergic manifestations elsewhere, by a careful history and sometimes by the occurrence of eosinophils.

In 1937, Dean[5] questioned a large number of patients with allergic rhinitis regarding ear symptoms and found that approximately 20% of those questioned did have ear symptoms. He divided these patients into four groups depending on the symptom syndrome which they presented. Group 1 was designated as eighth nerve crises, Ménière's symptoms without deafness. Group 2, typical Ménière's syndrome. Group 3, the largest group, was characterized by decrease in hearing with positive Rinné, sensation of fullness and deep itching in the ears; and in some instances vertigo, nausea and vomiting. Group 4 was characterized by migraine headache and ear symptoms. Dean noted that all patients with Ménière's symptoms suffered from a very severe type of allergy. In this same report he described one case of chronic otitis media in a patient with allergic rhinitis in which the ear became dry only after allergic control was established.

In 1943, Kuhn[6] made the statement that there is an appreciable number of patients with lowered hearing due to allergy. He listed as prominent symptoms in these patients: (1) Fullness of one or both ears; (2) loss of hearing or dullness of hearing; (3) deep dull pain in the ear; (4) itching in the back of the nose and between the

nose and ear; (5) tinnitus, vertigo or nausea; (6) tightness and drawing in the ear; (7) dull burning in the ear. He stressed the fact that hearing and other symptoms vary rapidly from hour to hour, day to day, and year to year, and states that the presence or absence of allergy can be immediately determined by making an audiogram, injecting adrenalin and repeating the test in 20 minutes. Marked improvement following the injection, he believes, is diagnostic of allergy.

In 1942, Chen Yue Mao[7] skin tested 535 deafened children inmates of an institution for the hard of hearing. Fifty-three per cent were biologically deafened, 47% were pathologically deafened. Of the 53%, 28.6% gave positive skin tests to one or more allergens. Of the 47%, 28.5% gave positive skin tests to one or more allergens. A very large group of normal children, on the other hand, showed only 2% clinical evidence of allergy. From his investigations Mao believes that it is not unreasonable to assume that allergy and deafness in combination have some mutual hereditary background.

A great many observers have reported deafness in cases of Ménière's disease in which allergy was suspected of being the etiologic trigger mechanism which set off the chain of inner ear symptoms. Notable among these observers are Duke,[8] Kobrak,[9] Portmann,[10] Dederding,[11] Furstenberg,[12] Wasowski,[13] Rowe,[14] Hansel,[15] Fowler,[16] Dean,[5] and more recently Jones,[17] Shambaugh,[18] Grove,[19] Kopetzky,[20] and others. All of these reports are of acute inner ear episodes and in most cases the etiology was thought due to sensitivity to foods. In none of these reported cases is there mention of any cytologic studies for eosinophils in the nasal mucus having been made either during or following the acute attack.

Amberg and Hewitt,[21] Cutter,[22] and MacCready[23] report one case each in which deafness followed the injection of tetanus antitoxin; the question of allergy as the etiologic factor was considered in each. Again no eosinophilic studies were made.

Even though little attention has been focused on allergy as it occurs in the middle and inner ear, a great deal of investigative work has been done on allergy of the nose, sinuses and upper respiratory tract.

Eosinophilic Leukocytes as Indicators of Allergy. Eyermann[24] in 1927 was the first to call attention to the importance of increased eosinophils in secretions as proof of nasal affection being of allergic nature. His work has been verified by Hansel,[25, 26] Finch,[27] Urfer,[28] Mullin and Ball[29], Leicher,[30] Shambaugh,[31] Walsh and Lindsay,[32]

Rachmann and Tobey, [33] Kallos,[34] Cowie and Jiminez,[35] Hume[36] and many others.

Hansel[37] has repeatedly stressed the fact that increased eosinophils or eosinophils occurring in groups in the nasal mucus is diagnostic of nasal allergy.

The presence of eosinophilic infiltration into any affected tissues during an allergic attack has been so constant that it is now recognized as one of the cardinal findings in allergy, and increase of eosinophils locally is now considered diagnostic of allergy.

Presence of Eosinophils in Ear Secretions. Hansel[37] in his book published in 1936, states, "In allergic individuals it is quite impossible to make a diagnosis of allergic otitis media unless marked predominance of eosinophils is demonstrated in the aural discharge."

He further states, "The involvement of the middle ear with an allergic lesion comparable to that seen in the nose characterized by edema and eosinophilic infiltration and eosinophils in the discharge has not been reported as yet. We have made a careful examination of all cases of acute and chronic otitis media and have not found a single instance in which a diagnosis of allergy could be established."

In 1942, Dohlman,[1] on the other hand, reported that many of his cases of chronic otitis which failed to become dry under all types of treatment were allergic in character. He arrived at this opinion by demonstrating increased numbers of eosinophils in secretions taken from the middle ear. He studied 178 cases (224 smears) and 99 showed eosinophils in percentages ranging from 9% to 40%. His diagnoses were further verified by history of other allergic mainfestations both in the patient and in the patient's family. All were skin-positive to one or more allergens.

Dohlman's findings were entirely contrary to Hansel's experience who, as already stated, examined similar secretions but was unable in a single instance to find eosinophils in sufficient numbers to prove a diagnosis of allergy.

Evidence Tending to Prove that Allergy Does Occur in the Middle and Inner Ear. In 1936, Dean[5] made the statement that "otitis media is a common complication of allergic rhinitis. The common finding in allergic rhinitis is a very edematous pharyngeal tubal cushion. It is only reasonable to suspect that there will be blockage of the tube and resulting changes in the tympanic cavity. We have found an increase in eosinophils in material taken 1 cm. up from the tube orifice, so we think we may assume that the

pharyngeal end of the tube at least, enters into the allergic process. We have not proven the tympanic end of the tube and the tympanum itself are *not* involved in it."

In 1939, Jones[17] stated, "Although scientific proof is lacking, clinical evidence indicates that the phenomenon of allergy does affect the eustachian tube as well as the middle and internal ear." He further observed that, "By means of a nasopharyngoscopic examination we may prove that the visible part of the eustachian tube is subject to the same objective changes which have been called characteristic of allergic changes in other nasal membranes. It is improbable that these recognized changes in the major membranes of the respiratory process and the eyes would not also affect the membranes of other extensions from the upper respiratory system, that is the eustachian tube and the tympanic cavity."

In his recent book "Deafness, Tinnitus and Vertigo", Kopetzky[20] states, "The local effects of allergy are not limited to the mucous membrane of the nose alone. The mucosa of the tympanic cavity and the lining membrane of the eustachian tube are also involved. Left untreated, these repeated attacks of allergy cause hyperplastic changes in the mucosa of the middle ear, which impede the transmission of sound wave impulses from the inner surface of the membrana tympani to the labyrinthian windows."

In 1947, Koch[2] published an important monograph entitled "Allergic Investigation of Chronic Otitis", in which he recorded his observations verifying Dohlman's findings that a high percentage of secretions taken from the middle ear in cases of chronic otitis media purulenta do show eosinophils in diagnostic numbers. Koch's experiments were planned, first, to prove whether or not a special clinical picture characterizes cases of chronic otitis with eosinophilic cell reaction in the secretions; second, whether or not it is possible in such cases to produce proof of an allergic etiology; and third, whether or not it is possible to experimentally elicit allergic otitis in guinea pigs.

His investigations included a study of 222 patients with 262 cases of manifest chronic otitis. Forty-one patients with 52 otites had a secretion picture of eosinophilic type. The remaining cases had no eosinophils in the secretion. The author characterizes the first type as eosinophilic and the second as aneosinophilic. Koch was able to determine a very definite microscopic difference between eosinophilic and aneosinophilic secretions and in most cases to predict beforehand what the microscopic findings would be. In the an-

eosinophilic type the secretion changed under treatment from purulent to mucoid and few eosinophils were present. In the eosinophilic type, however, eosinophils were present at the first or second examination or became apparent as the purulent discharge decreased and changed to mucoid secretion under local treatment with sulfathiazole.

Biopsies were made of the mucous membrane of the tympanic cavities of the same ears where perforations of the drum were present and section examinations showed eosinophilic infiltrations which ran parallel to the eosinophils found in the secretions from these same ears.

Experimental otitis which Koch was able to produce in guinea pigs by injecting horse serum into the middle ear of some and milk into the middle ear of others after previous sensitization to these proteins, showed pathological secretions full of eosinophils and marked eosinophilic infiltration into the edematous mucous membrane lining of the middle ear. Bacterial control cultures were sterile. His conclusions were, therefore, that the changes must have been due to allergic inflammation. His work was so comprehensive and his investigations so carefully controlled that his conclusions leave little doubt that allergic sensitization of the mucous membrane of the middle ear does occur.

Koch's experiments were outlined here in considerable detail because they furnish further scientific evidence supporting the contention that the middle ear does take part in allergic reactions similar to those seen in the nose and sinuses in rather a high percentage of cases and that allergy, therefore, must be considered one of the important factors in the etiology of certain types of deafness.

In 1937, Smirnov[38] injected guinea pigs previously sensitized to horse serum, with small amounts of the same serum into the middle ear on one side, while bouillon was injected into the other ear as a control. After a short time an intracardial injection of virulent streptococci was made. Growth of streptococci was noted in the ear which had been injected with serum, but not in the control ear.

This would seem to indicate increased vascular permeability, i. e., local allergic reaction in the serum-injected ear.

The opinions of the authors quoted, the brilliant and convincing work of Dohlman and Koch proving the presence of eosinophils in the middle ear cavity in allergic individuals with ear

symptoms, Smirnov's experiments, abundant clinical evidence and the fact that the mucous membrane of the eustachian tube and middle ear is continuous with that of the nose, sinuses and naso-pharynx all point to the premise that allergy does affect the eustachian tube and the middle ear.

It is logical, then, to assume that all parts of the mucous membrane lining the nose, nasopharynx, eustachian tube and middle ear cavity are susceptible to the same insults and diseases.

It also seems reasonable to suppose that the diagnosis of nasal allergy could be extended to include the nasopharynx, eustachian tube and middle ear.

Successful treatment of allergic manifestations of the nose and sinuses should be of great value in controlling acute ear symptoms due to allergy and in preventing permanent damage to the middle and inner ear.

It is quite possible that failure to recognize allergy of the respiratory tract as well as allergy of the middle and inner ear, and failure to institute general allergic therapy early in these diseases may explain at least some of the many failures which occur following our present day methods of treating potential deafness.

It should be emphasized that the two most important points in confirming a diagnosis of allergy of the nose and sinuses are (1) an adequate allergic history; (2) studies of nasal and nasopharyngeal secretions for increased eosinophils. These two important diagnostic points should always be investigated in all cases suspected of middle or inner ear allergy, and allergy should always be suspected in all cases of middle and inner ear episodes until it is ruled out as an etiologic factor.

COMMENTS AND CONCLUSIONS

1. Allergy as an etiologic factor in deafness is far more prevalent than is recognized at present.

2. Little attention has been paid to this phase of the problem and few studies have been made.

3. From the evidence here presented, it is logical to assume that the same allergic changes take place in the eustachian tube and middle ear as take place in the nose and paranasal sinuses, and that acute and chronic allergic episodes may effect both the middle and inner ear.

4. Any treatment aimed at relief of allergic symptoms of the respiratory tract should benefit allergic ear symptoms.

5. Allergy should always be suspected in all middle and inner ear affections until it is ruled out as an etiologic factor.

6. Examination for eosinophils in the nasal mucus is a most valuable test in confirming a diagnosis of nasal allergy.

7. Studies of biopsy specimens of pharyngeal and nasopharyngeal lymphoid tissue for increased eosinophils is also an important diagnostic procedure.

8. Both of these tests have been greatly neglected because of lack of interest on the part of most otologists in making such examinations.

9. Increased number of eosinophils in the nasal secretion or in the nasopharyngeal lympoid tissue in the presence of ear symptoms is presumptive evidence that the ear symptoms are due to an allergic episode.

10. An allergic nasal or sinal mucous membrane is frequently the media upon which infection is superimposed. In all probability the same is true of the mucous membrane of the middle ear.

11. Cytologic tests have shown that a very high percentage of all children with nasal symptoms are allergic. Many may have hearing defects.

12. Inhalants and food sensitivities are frequently responsible for irreversible tissue reactions in children as exemplified by lymphoid hypertrophies so frequently seen on the pharyngeal wall and in the nasopharynx.

13. A complete allergic investigation including cytologic examination of the nasal and nasopharyngeal secretions should always be made before x-ray or radium therapy in the nasopharynx is contemplated.

14. In a careful search of the literature, I have been unable to find mention of a single instance in which allergy has been adequately ruled out before irradiation was begun.

15. It is my opinion that local radium therapy to the nasopharynx is not the treatment of choice in deafness due to allergy of the eustachian tube and middle ear.

16. Hearing tests on a large group of proven allergic individuals have not been reported. Such studies repeated at intervals would do much to clarify the relationship between allergy and certain types of deafness.

17. It is believed that early treatment of allergic manifestations in the infant and young child will not only prevent much asthma and asthmatic bronchitis but will also prevent much adhesive deafness of later life.

18. The prevention of deafness due to allergy, then, is mainly a matter of early prophylaxis. If the allergy is not recognized and controlled before nerve damage has occurred or before irreversible tissue changes have developed in the eustachian tube and middle ear, the damage is permanent. Treatment then becomes limited to prevention of the further progress of the disease.

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DISCUSSION

Dr. James M. Robb (Detroit, Mich.): I want to say one thing about terminology. I don't believe there is any such thing as a simple mastoidectomy. About 25 years ago I did a mastoid operation on a young woman, and the mother heard that she had a simple mastoidectomy. In about ten days the girl died of meningitis. The mother said to me, "Doctor, that was not a very simple mastoid."

Through the last few years I have thought that some other term that the patient would not understand would be advisable and have used the term "cortical mastoidectomy," which I believe is used by the British.

Dr. Theodore E. Walsh (St. Louis.): There are two points that I wanted to bring up. The first is in regard to allergies of the ear.

There is no question that allergy plays an important part in impairment of hearing and deafness. Dr. Jerome Hilger of St. Paul gave a valuable presentation at the Academy on allergy of the ear but did not call it "allergy." He spoke of "vasomotor change" and I believe his term may be better.

One thing which bothers me when people speak of "allergy" is the thought that they think only of sensitivities to foreign proteins. I don't believe it is that simple. I feel all the work which has been done in allergy so far has accomplished relatively little. We are doing a great many skin tests, food eliminations, desensitization treatments, etc., but there has been no fundamental work done in what is the basic change that is causing these sensitivities. I make a plea that we think from a broader viewpoint not only of desensitizations, etc., but of what is the fundamental change in the organism as a whole, whether endocrine, or metabolic, or some other factor.

I wanted to ask Dr. Meltzer one question. He was speaking of modified radical mastoidectomies, and the hearing that is left after the ossicular chain has been broken and the incus and malleus removed. There has been a question brought up about the effect of the stapedius muscle in holding the stapes fixed after such a procedure has been done. Has he any information about the difference in hearing when the stapedius tendon has been cut?

I think it was Geoffry Bateman who spoke about it, or one of his men. I would be very interested to know what was the answer to that particular question.

Dr. Norton Canfield (New Haven, Conn.): I would like to ask Dr. Ashley if he has had an opportunity in his cases of allergy to do any blood study work such as has recently been reported by Dr. Kopetzky? Also has he done any other similar examinations that might give us some clue to that so-called allergy?

I feel, as Dr. Walsh does, the question of diagnosis has been inadequately worked out.

Dr. Edmund P. Fowler, Sr. (New York): Many children have deafness because of blockage of the eustachian tube due to allergy. When the allergy is better the tube opens and the hearing comes back. While sometimes the very mild allergies seem to cause a great deal of trouble, the most severe allergics often cause no trouble as far as the hearing goes. I would like to know why?

One of the greatest problems we have in the treatment of these children is the treatment of the parents. We should have more classes for parents. That is a very important supplementary measure in the training of these children.

There seems to be a tendency with the drug treatment of middle ear disease to neglect too often the mastoid operation. There is no one surgical procedure that was more important in the prevention of deafness than what we called the simple mastoid operation.

Dr. William E. Grove (Milwaukee): I was particularly interested in one remark that Miss Whitehurst made in reference to one of those youngsters, that the child was being prepared to be sent into a hearing school.

I think one of the most important objectives with these youngsters is to implement their education in such a way that they can be taken out of any school for specialized training for the hard of hearing and the deaf and be returned as soon as possible to the regular public schools.

DR. HENRY L. WILLIAMS (Rochester, Minn.): I believe Dr. Ashley has given us a most important paper. His subject is more fundamental than any of the others considered in this symposium. Allergy is associated with hyperplastic and hypertrophic changes in the adenoid tissues and in the nasal and sinal mucosa. Sinus disease frequently appears to be in large part secondary to allergic edema. The only thing that I can disagree with him on in the entire paper is his insistance on the importance of cytological studies of the nasal secretions. It seems so much easier to me to give the patient a test dose of one of the antihistamine drugs, after which, if he responds favorably, further tests are unnecessary.

I feel the confusion in some minds regarding the management of allergy is due to the fact that there are essentially three related types of allergy. All allergy is essentially a type of vasculitis. An allergic wheal cannot occur without a blood vessel being present, the permeability of which is increased to produce the typical allergic phenomenon. Therefore, environmental agents such as heat, cold, fatigue and emotional perturbation can also produce the clinical picture of allergy.

If we base our concept of allergy on the clinical picture and realize that this clinical picture can be produced by agents other than an antigen-antibody reaction, then I think many of the difficulties that we have had in the diagnosis and treatment of allergy will disappear. I have suggested that the basic form of allergy, physical allergy, is a stereotyped reaction of the smaller vessels, the arterioles and capillaries, to various environmental stresses. In later phylogenetic development localized tissue hyperimmunity developed, grafted on to the basic reaction. Then, almost as a by-product of tissue immunity, humoral immunity with its circulating antibody developed.

The hypothesis of allergy was developed by von Pirquet from his own observations and those of Ehrlich, Pfeiffer, Arthus, and others to explain certain observed clinical phenomena. Although Duke observed 20 years ago that the majority of clinical allergies were not explainable on the basis of this hypothesis, certain immunologists have continued to insist that, by definition, unless circulating antibodies are present the condition cannot be allergy. In other words, a clinical condition is observed which is termed allergy. This hypothesis was constructed to explain the occurrence of the condition. The majority of observed clinical cases do not meet the criteria established in the hypothesis. Instead of at once admitting that there must be error in the fundamental assumptions, certain immunologists wish to limit the term "allergy" to those few cases that do meet the criteria established by the hypothesis. I am unable to follow this kind of reasoning if it can be dignified by such a term.

Can we wonder at our failures in the treatment of allergic cases by hyposensitization when, if we exclude inhalant allergies, especially pollenosis, from consideration, less than 10% of the allergies seen in connection with otologic or rhinologic practice can be demonstrated to have either circulating or fixed specific antibodies present?

Dr. Ashley is to be commended particularly for calling to our attention the fundamental role that allergy so often plays in infections about the ears, the nose and the throat. This role cannot be too frequently or too emphatically expressed.

I would enter a plea, however, that we do not concern ourselves exclusively with the injection of antigenic material in treating such patients but rather consider therapy on a broader basis and return to the medical treatment of allergy.

DR. DOUGLAS MACFARLAN (Philadelphia): I want to compliment Miss Whitehurst for her presentation. It is the kind of presentation that I wish there were more time for. There are several things she could not cover, some of which I must stress as they affect the otologist.

When these children are first brought to us, they are not like the four you have seen. They flit from one thing to another, have temper tantrums, are an attention problem, and behavior problem. These are the first difficulties to get over. The children shown by Miss Whitehurst are now past that stage.

When first seen these children are attention-deaf. Their mothers and fathers may say that they do not show attention to sounds, and if you infer from that statement that they have no residual hearing, you are apt to refer the case to a school for the deaf.

When you test them and train them, you find that 80% of them have reachable and useable residual hearing even though at first they are unresponsive. A hearing aid is put on all of them, even those aged two years. It is surprising the results you can get. By auricular training these children are ready at five or six to go into grade school with normal children where they are in a normal environment as well as maintaining home contacts. Much depends upon the training of the parents in the handling of the education. The follow-up must be strict and painstaking.

DR. GORDON D. HOOPLE (Syracuse, N. Y.): I am surprised in a discussion which includes all the ground that has been covered in this one that several have not spoken on the matter of irradiation.

I want to put on record my personal experience. Before leaving for my war service, I did not make available to myself a radium applicator. I was away from this country three years at the height of the popularity of the use of the radium applicator. I came back to find that most of my fellow laryngologists were using it frequently. I used it fairly frequently myself on my return from the service. I have been back a little over three years and am now quite surprised at the infrequency with which I find indications for its use when I have otherwise properly treated the patient.

DR. DONALD F. PROCTOR (Baltimore): I agree with Dr. Ashley that the role of allergy is very important though an infrequent complication, and we have considered it in our cases. We have found in many instances that children not responding to desensitization therapy have been benefited by the addition of irradiation of infected adenoid tissue. The allergists practicing in Baltimore feel it is a great help in many instances.

I myself have removed adenoids after irradiation and sometimes have been discouraged to find how much tissue remains. Certainly irradiation is not the answer in every case. We could use very large doses of radium and nearly all the patients would respond satisfactorily; but with the small dose only a fair percentage of them respond, probably 70-75%. Such dangerously large doses are obviously not justifiable.

Radium does have one advantage over adenoidectomy in those patients with relatively small amounts of adenoid tissue in that it is so simple. It does not require bringing the patient into the hospital and giving him a general anesthetic. We feel that in our hands when there is only a small amount of adenoid tissue, irradiation is preferable to surgery.

DR. REA E. ASHLEY (San Francisco): I will comment on Dr. Walsh's discussion first. We do not consider skin tests of much value in diagnosing perennial allergy. We depend mainly on the history in making our diagnosis. If we find a youngster who has "frequent colds" even after tonsils and adenoids have been completely removed and that child still has a persistent cough at night—and the percentage of these patients is not small—ninety-nine times out of a hundred the child is allergic. I do not agree with Dr. Walsh that very little has been accomplished in the treatment of these patients. I do think, however, that very little has been done about allergy by the otologists and by the majority of rhinologists. The usual routine is to send these patients to the allergist who usually has little interest in what changes take place in the nose, throat or ear and that is the

last the otologist sees of the patient. I believe that until the otologists and the rhinologists can be sold the idea of doing their own allergy diagnosis and treatment so that they can become "allergy conscious" and competent to recognize allergy, they will not get very far in the treatment of these conditions.

Dr. Canfield has asked if we had any experience with blood studies. They are very difficult to do and expensive to the patient. We have had them in mind and have obtained complete blood studies in a few patients. However, from these studies we were unable to arrive at any definite conclusions. The whole subject of allergy is so tremendous that it not only includes blood studies but also nutritional difficulties, glandular abnormalities and many other factors.

Dr. Fowler, I did not mean to leave the impression that I believe allergy of the lower end of the eustachian tube to be the only important factor. In my opinion, allergy affects not only one tube or one side of the nose but the entire patient. However, one side of the nose may be affected and the other not, or one ear may be affected and the other not, regardless of where the original sensitization took place.

In today's discussion I had in mind particularly the allergy due to protein sensitization and not physical allergy which was mentioned by Dr. Williams. Dr. Williams does not believe in cytological studies but these are the only positive laboratory tests we have and they have proved of value in our practice. The tests are simple to do and if negative should be repeated several times. Eosinophils disappear in the presence of acute or chronic infection. We have been successful in making a diagnosis of allergy even when it is in combination with infection by giving the patient injections of penicillin for two or three days and then taking a smear from the nasal mucosa. Frequently we find eosinophils in diagnostic numbers which were completely hidden by polymorphonuclear leucocytes before the penicillin was given.

I can not agree with Dr. Proctor that allergy does not play a large role in many of these ear conditions. There are many cases of so-called infection of the eustachian tube and middle ear in which allergy is the basic factor. If examinations are made after the infection has subsided, a surprisingly large percentage of the cases will prove to be allergic.

I should once again like to emphasize that I believe the members of our specialties will have little success in obtaining results in allergic patients until they take over the allergic management themselves.

Dr. Meltzer (Boston): I believe in the use of radium, roentgen ray and in direct adenoidectomy as measures to be used for the eradication of lymphoid hypertrophies in the nasopharynx and around the eustachian tube. The important thing is to exercise the best possible judgment as to when each of these measures is to be used.

I am always impressed by the vast number of children who have had tonsillectomy and adenoidectomy performed by general practitioners, young internes and general surgeons, men not particularly trained in this procedure; when these children are examined by me for various reasons, I find in the majority of cases an excellent, clean nasopharynx. This indicates to me that lymphoid tissue is a regressive structure and we must keep this in mind and not be too hasty in recommending treatment. We have all had children whom we treated once, twice, or more times a year during their childhood who had either tubal closure or otitis media in the period before radium or x-ray was used, and I have observed that as time went on these children have not come to see me for this complaint. Their hearing is normal, or possibly they have a 10-db loss in the higher frequencies, their nasopharynx is clean and all lymphoid tissue has disappeared. If a simple inflation with a Politzer bag restores the hearing to normal in these children with an occasional secretory otitis following upper respiratory infection, I believe they should be left alone unless the adenoid is really of such size that it should be removed. I will definitely agree that if the lymphoid tissue is flat and these tubal

closures recur with such frequency that the hearing fails to return to normal with a simple inflation, that radium, or roentgen therapy is definitely indicated.

I have yet to see shrinkage of lymphoid tissue of the lateral bands that run along the posterior pillars of the tonsils, by either roentgen ray or radium. I have tried hard to see this resolution of lymphoid tissue after radium therapy but I have failed to see it. Associates in my department have said to me, "Look at the effect of radium on this adenoid tissue—see how it flattened it out." Later the same patient would come in with an acute nasopharyngitis and I have asked the same group, "Please look at the nasopharynx now." What had been an insignificant amount of lymphoid was now half the size of a raspberry. That is what this tissue is capable of doing under certain circumstances and that is why I prefer direct adenoidectomy to any other procedure if the tissue is sizable.

I am not able to answer Dr. Walsh's question. The physics involved is too intricate for me to explain the effect of cutting the stapedius tendon. Dr. Glen Wever wrote a very illuminating paper on the microphonic response when the ossicular chain is interrupted in animals. I believe the loss would range between 40 and 50 db.

The question has been asked about conservation of hearing in the modified radical mastoidectomy. I can generally retain hearing by the techniques described. I might lose 5 or 10 db where the ossicular chain has to be interrupted, but where the stapes is movable in an air-contained space, I am able to maintain hearing to a serviceable degree.

MISS MARY W. WHITEHURST (New York): The problem of behavior and attention in these children is a very definite one but that can be licked in the very beginning. I try to pass the job on to the mothers as much as possible. I cannot work with children who have tantrums in the office and I feel that the mother has to be oftentimes educated to working with her own child at home because it is not formal education for a little two-year-old or three-year-old. It is the mother's job. A lot of my work is actually working with the mothers trying to show them how to work with the very young until they co-operate sufficiently to come to the office for some more or less formal training.

Clinical Notes

LXXVIII

BILATERAL CHOANAL ATRESIA

REPORT OF A CASE IN AN INFANT AND

REVIEW OF LITERATURE

RICHARD W. HANCKEL, M.D.

CHARLESTON, S. C.

In recent years, reports on congenital choanal atresia have become increasingly more frequent, because of the fact that many cases have been detected during routine examination of patients in the armed services.

This anomaly is rather rare, and was first commented on by Otto[2] in 1829, when he observed it at autopsy. Since that time many reports have appeared in the literature, so that by 1910 Fraser[3] estimated the number to be 117. Cinelli[4] in 1940 gave the total number as 220. Boyd[5] in 1945 was able to account for 230 cases. Further examination of the literature by the author shows the total at the present time to be 235.

There are several theories as to the development of congenital atresia and there are several varieties of the condition itself. Each type of developmental anomaly produces a different type of the final condition as follows:

1. The persistence of the nasobuccal membrane (see Fig. 1). In this case the membrane is 1-2 mm within the choanal cavity and consists of mucous membrane and varying amounts of bone. The reason for this is that the epithelium, growing inward to form first the nasal pit or pouch then the nasal sac, meets the buccal epithelium at this point. Between the two layers of epithelium lie varying amounts of mesodermal tissue. Normally the mesodermal tissue is absorbed and the epithelial membranes become attenuated and rupture spontaneously on the thirty-fifth to thirty-eighth day of fetal life. Failure of this change results in the persistence of an obstructive membrane, complete or partial (a small central perforation may
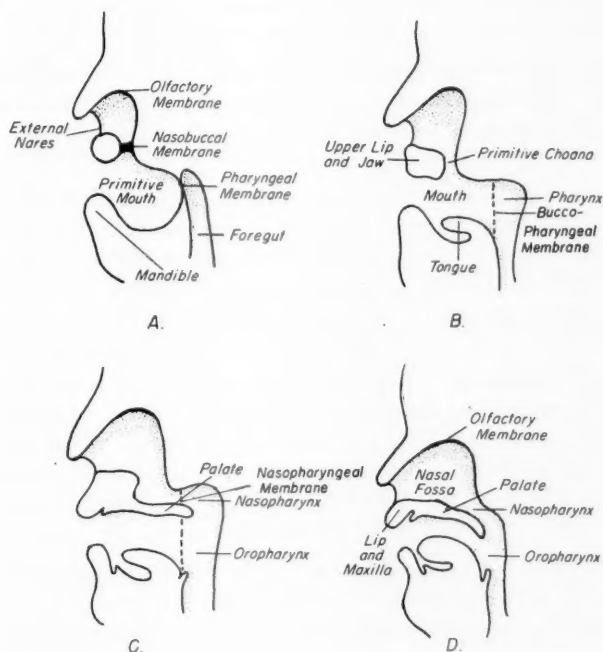
Fig. 1.—Schematic representations of the embryologic development of the nasal, oral and pharyngeal cavities, with the choanae. Reprinted from Dr. H. M. E. Boyd[5] after Dr. B. C. Colver.

occur), containing variable amounts of bone between the layers. The bone is derived from the unabsorbed mesodermal tissue. This type contains no cartilage because the bone from this mesodermal tissue is not first laid down in cartilage.

2. A medial overgrowth of the vertical and the horizontal palatal processes. These palatal bones are laid down first in cartilage, consequently these obstructing choanal membranes may contain bone and cartilage between the layers of epithelium. They occupy a like position to those under type 1, that is, 1-2 mm within the nasal cavity.

3. The persistence of the nasopharyngeal membrane. As the foregut pushes upward it is separated first from the buccal cavity and then from the nasal cavity by the buccopharyngeal, then the nasopharyngeal, membrane respectively (see Fig. 1). Failure of this membrane to rupture spontaneously results in the formation of a

membranous obstruction containing neither bone nor cartilage and attached to the most posterior part of the choanal boundary.

These are all theories on an embryological basis as advanced by Stewart.[1] Thus congenital atresia may be classified as membranous, membrano-osseous, or mixed. It may be partial or complete, and either unilateral or bilateral.

The boundaries of the choana are as follows: Laterally, the medial plate of the pterygoid process of the sphenoid bone and the perpendicular plate of the palatal bone; inferiorly, the horizontal plate of the palatal bone; medially, the posterior border of the vomer and the nasal crest of the palatal bone; superiorly, the ala of the vomer and the body of the sphenoid bone.

In the newborn, bilateral choanal atresia is a medical emergency, because of the obstruction to breathing and the accompanying feeding problem. Dyspnea results because the child has not as yet learned to breathe through the mouth. Attempts at nursing make dyspnea much more marked. This atresia may be the unsuspected cause of death in many infants.

Unilateral choanal atresia presents no emergency problem and indeed may pass unnoticed for many years.

Further indications of the presence of the condition are as follows: The presence of a tenacious mucus in the affected nostril or nostrils; obstruction to passage of a probe through the occluded side; x-ray evidence of the inability of iodized oil to pass through the nostril into the nasopharynx.

If the condition is undiagnosed in infancy or early childhood, other signs and symptoms may be: History of inability to breathe through the affected side; history of a feeding problem in infancy; a high, arched palate on the occluded side or on both sides if bilateral. The patient is a mouth-breather (if bilateral) and the upper incisors may be irregular. Anosmia is usually present. The nasal septum is usually deviated to the side of the atresia if it is unilateral. Examination with a postnasal mirror and nasopharyngoscope presents typical appearances. Adenoids are usually not present, nor is sinusitis a complicating factor. Involvement of the middle ear is unusual.

Occasionally one finds other congenital anomalies associated with choanal atresia (the author's case also had polydactylism). Heredity apparently does not play an important part in choanal atresia.

Because of the rarity of the condition, one man rarely sees more than one or two cases during a lifetime of practice and therefore the

types of treatment have run the gamut of the imaginations of those who have encountered cases. There is no generally accepted uniform surgical procedure for the treatment of this condition.

A review of the types of treatment developed since this condition first became known in 1839 shows that they may be grouped under several heads. A résumé of these follows:

1. *Surgical Intranasal Approach*:

    a. Emmert[6] in 1853 used a curved trocar.

    b. Hubbel[7] in 1886 used a hand drill.

    c. Clarke[8] in 1898 and Pfingst in 1914 used both electric and hand trephines.

    d. Katz[9] in 1911 and Pfingst in 1914 made drill holes through the atretic wall and enlarged these with a saw and biting forceps.

    e. Katz[9] in 1911 and White[10] in 1919, and earlier perhaps Mackenty, advocated removal of the posterior quarter of the nasal septum as the final step in operation.

2. *Cauterization of the Offending Membranes*:

    a. Von Schroetter[11] in 1885 successfully used electrocoagulation.

    b. Various chemical coagulants have been used unsuccessfully.

    c. Morgenstern[12] in 1940 advocated electrocoagulation where the atretic structure is entirely membranous or the bone is very thin.

    d. Bourgeois and Leroux[13] in 1933 used electrocoagulation with olive tip electrodes on thin membranes.

3. *The Transseptal Approach*:

    a. Uffenorde[14] in 1908 first used curettes and chisels via the nasal and septal roofs.

    b. Richardson[15] in 1913 advocated a complete submucous resection as a preliminary step.

    c. Stewart in 1931 also did a complete submucous resection as a preliminary step.

    d. Kazanjian[16] in 1942 divided the columella close to the floor of the nose, extended the incision posteriorly to the posterior border of the cartilaginous septum, then upward. The anterior septum was then turned up and out of the way and
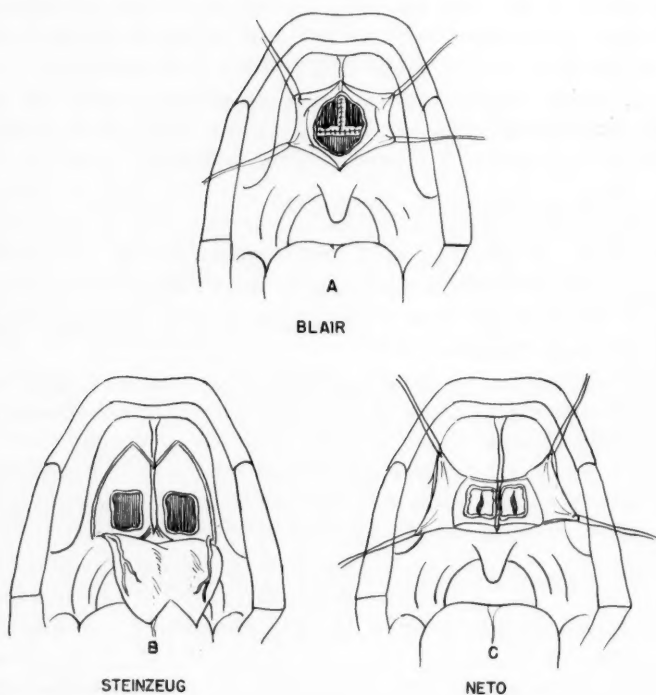
Fig. 2.

the bony septum and occluding membrane entirely removed. The cartilaginous septum was then turned down and sutured into place.

    e.   Roberts[17] in 1944 removed the bony occlusion via the transseptal route in an adult.

4.   *The Transpalatal Approach*:

    a.   Brunk[18] in 1909 used a midline incision.

    b.   Blair[19] in 1931 also used a midline incision and removed most of the hard palate, bony and cartilaginous septum, and the occluding membrane (see Fig. 2 A).

    c.   Steinzeug[20] in 1933 used Awerbuch's approach with success (see Fig. 2 B).

    d.   Schweckeniek[21] in 1937 used the transpalatal approach on two infants, one ten days old and the other nineteen days
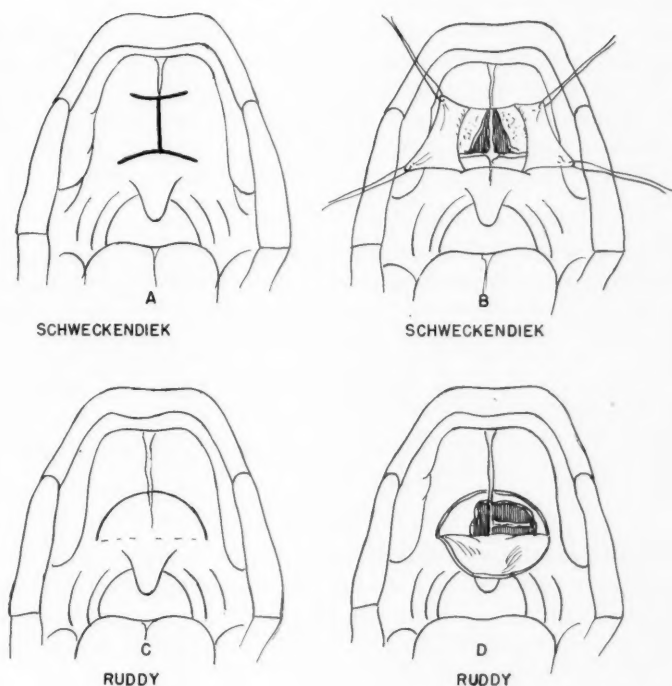
Fig. 3.

old. In one the palatal flaps were sutured in place and subsequently sloughed. Through the opening he was able to see the beginning formation of a fibrous wall. This he removed by curettage and diathermy. Bouginage was used sparingly as he was of the opinion that it was apt to produce shock. In the other case he did not suture the flaps. Results in both cases were satisfactory (see Fig. 3, A and B).

e. Neto[22] in 1942 used a transpalatine approach similar to Schweckendiek's and with some modification of the aftercare. His results, too, were satisfactory (see Fig. 2 C).

f. Ruddy[23] in 1944 used a U-shaped incision on a three-year-old child with unilateral choanal atresia with satisfactory results. (This same technique was used in the author's case (see Fig. 3, C and D).

5. *Combined Approach*:

a. Klaff[24] in 1944 split the uvula and soft palate in the midline, retracted each side with traction sutures and, working under direct vision and using his finger as a guide, broke down the occluding membrane with an antral rasp intranasally. The wound was closed and a large catheter left in place for ten days. The opening was patent a year later.

6. *Transantral Approach*:

a. Wright, Shambaugh and Green[25] used the transantral approach on an adult under block anesthesia. This was reported in 1947. In their opinion the advantages of this approach are:

(1) Better exposure of the operative field.

(2) Ready control of hemorrhage, if it occurs, by ligation of the internal maxillary artery.

(3) Less danger of injuring the pterygopalatine nerves and blood vessels.

COMMENT

Various types of materials for obturators ranging from tin to rubber have been used to maintain the patency at the operative site. Skin grafting has been used advantageously for this purpose. Also there is no contra-indication to the frequent use of bouginage as will be seen in the author's case.

The type of anesthetic used in these cases varied from local, to sedation with barbiturates, to inhalation with an ether hook.

There appears to be no optimum time for operation, each case being judged on its individual merits. Certainly there is no contra-indication to operating on very young infants. The age of patients who have been operated on varied from ten days of age to young adults, with equally good results.

The type of operative procedure used should be determined by the type of obstructing membrane, the age of the patient, and the condition of the septum.

If the occluding membrane is membranous or if bone is present but very thin and the patient an adult, the procedure of choice would be electrocoagulation after the method developed by Morgenstern,[12] using a local anesthetic.

If the membrane contains bone, or bone and cartilage, and the patient is an adult, the procedure of choice would be transseptal approach as advocated by Kazanjian[16] and Roberts,[17] using a local anesthetic.

If the patient is an infant or a child, the best procedure would be transpalatine approach because of the better exposure obtained and the larger area for manipulation of instruments. The anesthetic used may be local preceded by a barbiturate, or general, using ether by way of an ether hook, or given by tracheotomy tube if a preliminary tracheotomy has been done (as was necessary in the author's case).

REPORT OF A CASE

B. J. C., a white female infant, was admitted on March 11, 1947, a few hours after birth because of marked respiratory difficulty. This was a full-term infant, spontaneously delivered, who had marked difficulty starting to breathe at the time of birth. This difficulty continued as a major feature and the patient was transferred to the nursery at this hospital where an incubator was available.

On admission a large amount of yellow fluid was removed from the mouth by postural drainage and aspiration. The baby was cyanotic and breathing only in infrequent gasps.

On examination the following positive findings were noted:

1. Cyanosis.
2. Feeble respiratory effort.
3. Some overriding of sutures about the anterior fontanelles.
4. Poor aeration of lungs.
5. An extra digit on the outer side of both little fingers.

The impression of the first examiner was:

1. Cerebral birth injury.
2. Possible aspiration asphyxia.

It was stated that the patient was given oxygen by nasal catheter, and was placed in an incubator with her head down. Color and breathing improved.

The periods of dyspnea recurred whenever the baby was removed from the incubator. She had considerable difficulty in swallowing. Frequently after taking the formula the patient would vomit.

The extra digits were amputated on March 22, 1947, under local anesthesia.

On admission the weight was 6 lb. Seventeen days later, on March 28, 1947, the weight was 5 lb. 6 oz. On this latter date a
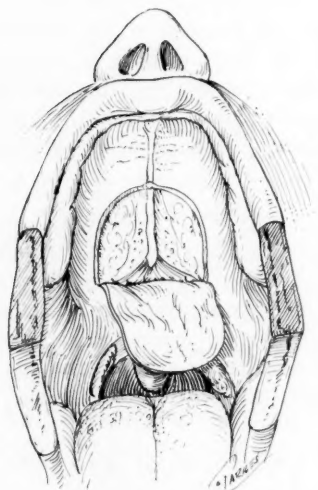
Fig. 4.

consultation of the otorhinolaryngology staff for a laryngeal exam-
ination was requested. Because of the poor condition of the patient
(she was still in an incubator), a very rapid direct examination was
done with a laryngoscope and no further examination attempted at
this time. A normal larynx was observed.

On the following day it was noted that a mucopurulent nasal
secretion was present and that both nostrils were apparently com-
pletely obstructed. An attempt was made to pass a cotton-tipped
applicator through the nostrils into the nasopharynx, but both
passages were found to be blocked posteriorly. Subsequently on
April 3, 1947, x-ray examination of the nasal passages after instill-
ing iodized oil confirmed the diagnosis of bilateral choanal atresia.

By April 10th, no dyspnea occurred on removing the patient
from the incubator except during feedings, when dyspnea and re-
gurgitation of food were frequent. The weight was 5 lb. 9 oz.
She was transferred from the incubator in the nursery to a crib in
the pediatric ward.

On the following day, April 11th, when the patient was one
month old, a tracheotomy was done to relieve the dyspnea at feed-
ings and allow the patient to gain weight and strength to withstand
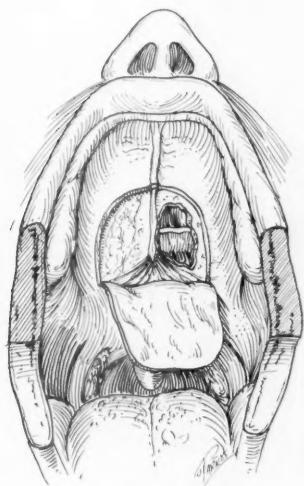the operation contemplated.

Fig. 5.

By April 21st the patient weighed 5 lb. 11 oz. and though she had not regained her birth weight of 6 lb., she appeared in good general condition and a transpalatal resection of the membrane obstructing the posterior choanae was done. The transpalatal approach was selected as the one of choice because of the small size of the patient.

Operative Technique: Ether vapor was given via a soft rubber catheter introduced in the tracheotomy tube. The patient took the anesthetic poorly throughout, having numerous periods of apnea.

The patient was arranged as for a cleft palate operation, with her head off the end of the table and resting on the operator's lap.

A U-shaped incision was made through the mucous membrane of the hard palate, the convexity being anterior and the "legs" being just medial to the posterior palatine vessels and nerves.

The mucoperiosteal flap was reflected posteriorly just beyond the posterior border of the hard palate. The mucous membrane was very thin over a ridge of bone in the midline and this was unintentionally torn longitudinally in the midline during the procedure.

The posterior border of the hard palate on the right was removed a distance of several millimeters up to the occluding membrane. A small opening was made in the hard palate just anterior to
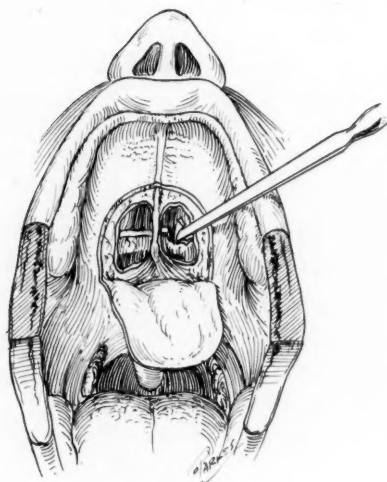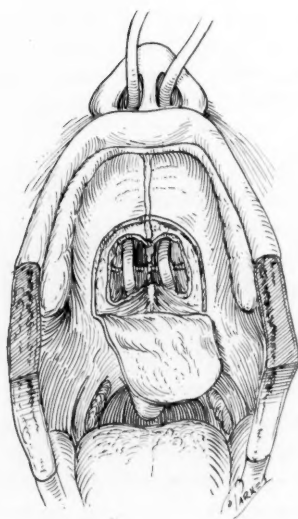
Fig. 6.

Fig. 7.

the occluding membrane. This was enlarged so that the complete inferior border of the occluding membrane was visible. The membrane was found to be thicker along this border than superiorly, and consisted of bone between the two layers of mucous membrane. The occluding membrane was removed with chisels and biting forceps.

A similar procedure was carried out on the left side and the left atretic membrane removed (see Fig. 4, 5 and 6).

This procedure left the posterior third of the nasal septum exposed in the midline and this, too, was removed. It was found to be cartilaginous at this stage of development.

Rubber catheters were passed through the nostrils into the nasopharynx and anchored with a black silk mattress suture through the columella (see Fig. 7).

The mucoperiosteal flap was sutured back in place with interrupted black sutures starting laterally and proceeding to the midline first on one side then the other. The tear in the midline was also repaired (see Fig. 8).

The patient's condition at the end of the operation appeared to be as good as it was at the beginning.

There was very little bleeding during the operation.

The pathologist's report showed only bone, no cartilage, in the specimens from the atretic membrane.

Postoperative Course: The patient had been given 10,000 units of penicillin every three hours intramuscularly for two days pre-operatively and this was continued until April 27th, six days after operation. A low-grade temperature rise up to 100.4° F. was noted for a few days immediately after operation.

By April 30th, nine days after operation, the weight was 6 lb., her birth weight.

On May 1st the catheters which were serving as obturators were removed from the nostrils.

After blocking the tracheotomy tube for 24 hours it was removed on the morning of May 3rd. Dyspnea during feeding in the afternoon necessitated re-insertion.

The incision in the midline of the palate had sloughed leaving a fistula between the mouth and nasal cavities. Through this dehiscence it was noted that granulation tissue was growing in to form another obstruction in the posterior nares. On May 23rd the granulations were removed with a mastoid curette under general
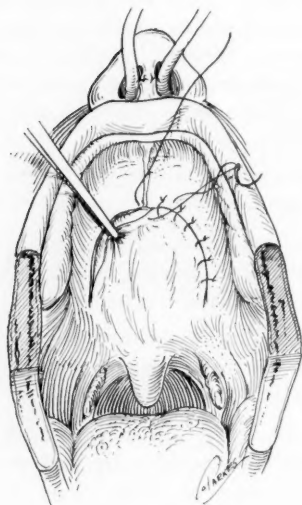
Fig. 8.

anesthesia in the operating room. A No.10 French catheter was passed into each nostril and sutured in place to the anterior part of the septum with a mattress suture of black silk as before. The uvula was amputated at this time also to see if this would facilitate the breathing.

The nasal catheters were left in place until June 16th, a period of about three weeks. During this time the temperature remained within normal limits. Penicillin, 20,000 units every three hours, was given intramuscularly from May 23rd to 27th, i.e., immediately pre- and postoperatively.

After removal of the catheters on June 16th, the posterior nares were dilated daily by passing a No. 10 French catheter through the nostrils. Despite this procedure the posterior nares again became obstructed so that by June 21st, five days later, it was impossible to pass a catheter into the nasopharynx.

On June 23rd the patient was again taken to the operating room and by retracting the lips of the fistula through the soft palate the granulations (not as abundant this time) were again removed. The catheters were re-inserted in the nostrils and sutured in place as before.

By July 2nd the baby's weight was 7 lb. 8 oz., the general condition was good, and though the tracheotomy tube and nasal catheters were still in place, it was decided to discharge the patient from the hospital. The patient was now taking her formula well and there was no dyspnea at feedings. She returned home on July 2nd.

The patient returned to the hospital at weekly intervals to have the tracheotomy tube changed. She apparently was doing well until August 1st when she was readmitted with acute upper respiratory infection and a temperature of 102.4° F. This promptly subsided on penicillin therapy, transfusions and general supportive therapy. Weight on admission was 8 lb. 1 oz.

On August 5th the nasal catheters were again removed, this time having been in situ for six weeks. The posterior nares were dilated several times a day by passing a No. 10 French catheter through the nostrils into the nasopharynx. This was continued throughout the hospital stay and at home by the mother after discharge from the hospital on August 12th.

The tracheotomy tube was blocked off on August 7th and finally removed on August 9th, approximately four months after its original insertion.

The patient has been checked at regular intervals since her return home, and at the last visit on September 11th the posterior nares were still patent, the tracheotomy wound was completely healed and the child weighed 10 lb. The fistula in the hard palate was almost completely healed and did not complicate the feedings. She was six months old at that time.

COMMENT

It is self-evident that in these cases a long period of hospitalization may be necessary and should be prepared for.

Instead of suturing the mucoperiosteal flap over the hard palate it would be just as well to leave this open as the flaps apparently usually slough anyhow, and if left open an adequate window is left to remove granulations which almost inevitably reform in patients so young.

It is the author's belief that if the patient's condition permits, it would be better to delay operation on the atretic structure until the patient reaches sufficient size to make the technical procedures at operation less difficult. In this case, in which it was necessary to do a tracheotomy in order to maintain an adequate airway, the author feels that the early removal of the choanal obstruction was justified.

Apparently there is no contra-indication to frequent bouginage to maintain patency of the posterior nares after operation.

The location of this obstruction and the fact that only bone was found in the specimen on pathological examination indicates that it probably developed as a result of the persistence of the naso-buccal membrane (type 1 in the author's classification).

SUMMARY

A review of the literature on congenital choanal atresia is presented and a classification of the various types is offered.

The types of operative procedures, as developed to date, are presented and suggestions made as to which may be used in the various typs of obstruction in adults and children.

A case report is presented giving the operative procedure and postoperative course in detail. Comment on the case is made.

96-A BULL STREET.

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LXXIX

THE RELATIONSHIP OF CHRONIC RECURRENT SIALADENITIS TO THE ALARM REACTION

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Recurrent sialadenitis is a clinical entity distinct from mumps, Boeck's sarcoid, suppurative parotitis, or obstructive parotitis. The condition may occur at any age and has a benign course. There is, however, no general agreement as to its etiology and the clinical picture seems confused.

Of the cases hitherto reported, many have been described in children.[1-4] In some of these cases there was often associated asthma, hay fever, urticaria, or angioneurotic edema, and also eosinophilia in the blood and at times in the parotid secretions, whereas in others there was no evidence of allergy. Ascending infection through Stensen's ducts was another commonly postulated cause. A biopsy of the parotid gland performed in one of the latter cases showed diffuse chronic inflammation and obstruction of the ducts by inflammatory exudate.[1]

Forty-four cases have been reported in adults.[1-11] In twelve of these, there were manifestations of allergy such as a history of hay fever, asthma, or urticaria, increased eosinophilic leukocyte count in the blood and occasionally eosinophilic leukocytes in the parotid saliva. Only in one of these allergic cases was purulent parotitis believed to be superimposed and in none was there sialodochectasis. In twenty-eight cases there was no evidence of allergy and these could be classified as purulent recurrent parotitis, often with sialodochectasis. Four cases were not studied in detail and cannot be classified. Two observers noted a high incidence of emotional instability including manifest psychoneurosis or even psychosis.[1, 6] We have observed two cases of recurrent sialadenitis in adults in which neither infectious nor allergic factors could be demonstrated.

REPORT OF CASES

CASE 1.*—The patient was a male Negro, aged 40, who was a painter. He gave a history of mumps at the age of 18 with left orchitis. He was in good health until November, 1942, when he was inducted into military service and engaged in maneuvers in the California-Arizona desert. Following this sudden change from his usual civilian activities and shortly after being alerted for overseas duty, he developed swelling of both sides of his face and a tremor of his hands. The swelling of the face subsided after a week, but subsequently he began to have frequent recrudescences. These swellings gradually subsided after his arrival in the European Theater of Operations.

In 1945 while riding in a truck in Germany, an 88-mm shell exploded nearby. He was thrown to the ground but did not lose consciousness. Shortly thereafter he had an acute recrudescence of his parotid gland swellings, this time more severe than before. Between 1943 and 1948, he had more than 50 recurrences, each lasting about one week and then subsiding. When examined in May, 1948, the swellings were nontender and no nodules were felt. Examination of the mouth was negative. Pressure over the parotid glands produced a flow of clear saliva from the ducts. No pus was obtained from either duct on any occasion. Examination of the parotid secretion did not reveal any eosinophils. Culture of the secretion yielded *Streptococcus viridans.* Blood counts were normal with no significant eosinophilia. Wassermann and Kahn tests were negative. Soft tissue roentgenograms of the face did not reveal any calculi in the maxillary or parotid regions. Sialography of the left parotid gland showed a normal configuration of the duct system. Coccidioidin skin test 1:100 was negative. Mantoux first strength PPD was 2+. X-ray examination of the chest, hands and feet were negative. Sedimentation rate varied between 18 and 23 mm (Westergren). Serum albumin, globulin and total protein were within normal limits. Psychiatric evaluation revealed a psychoneurosis.

CASE 2.—This 45-year-old white woman was seen originally in August, 1948, because of attacks of substernal pain for the previous six months. Thorough investigation at that time revealed no organic cause for the pain. Electrocardiogram, chest x-ray, urine, blood pressure and general physical examinations were negative. She

was found to have a low basal metabolic rate (-28%) which be-
came adequately controlled by small doses of thyroid.   She also
required estrogens for menopausal symptoms.   Psychiatric consulta-
tion revealed a profound emotional disturbance attributable to sex-
ual maladjustment in her marriage.   Five years previously, at the
age of 40, she was married for the first time to a man two years her
senior, also married for the first time.

The past history was not notable except that in 1944 a cyst
had been removed from her left breast.   In 1937 she had had mumps,
following which her menses ceased.

In October, 1947, she complained of a sore throat and noticed
rare purpuric spots.   Complete blood count including platelets was
normal.   Tourniquet test, bleeding time, clotting time and clot re-
traction were normal.   The infection failed to respond to sulfadia-
zine and penicillin, persisted for six weeks and then subsided.   Throat
culture revealed no significant pathogenic organisms.

In February, 1948, she developed a swelling in the region of
the right parotid gland associated with fever up to 101° F., slight
increase in the sedimentation rate up to 30 mm/hour (Westergren),
but no leukocytosis.   She also had occipital headache, some swelling
of the left parotid gland and of the submaxillary gland.   These
symptoms subsided in ten days and subsequent swelling usually in-
volved only one parotid gland, lasted several days and then sub-
sided for several days only to recur either on the same or opposite
side.   Between attacks of acute swelling the gland was felt to be
slightly large and tender.   For the subsequent year, attacks con-
tinued to recur.   The complement fixation test for mumps was
negative.   Blood counts were repeatedly normal without significant
eosinophilia.   The parotid secretion was always clear and contained
no pus or eosinophils.   A sialogram of the left parotid gland was
normal.   It is to be emphasized that this patient continued to be
subject to considerable emotional stress and that during the period of
parotid swelling, the chest symptoms seemed to subside.

DISCUSSION

Selye[12] and others have demonstrated organic changes in the
liver, spleen, thyroid and adrenal glands, lymph nodes, gastro-
intestinal tract and pancreas as manifestations of a nonspecific alarm
reaction.   Ehrich and Seifter[13] attempted to produce a similar reac-
tion in the parotid glands of rats because of their high content of
ribose nucleic acid in common with other structures involved in the
reaction.   They were successful in producing focal areas of severe
necrosis in the parotid glands.

Since the sialadenitis in the cases described here was found in emotionally unstable individuals subjected to severe social and environmental stress, it seemed possible because of the nonspecificity of the inflammation that our cases might be manifestations of the alarm reaction. Whether this factor is a unifying element in the other reported cases cannot be determined at this time.

SUMMARY

1. Recurrent sialadenitis has been attributed to allergic or infectious factors by most authors, although two observers have noted a high incidence of psychoneurosis in such patients.

2. Experimental work has shown that the parotid glands participate in the alarm reaction.

3. Two cases of recurrent sialadenitis are presented in which no allergic or infectious factors could be demonstrated.

4. The sialadenitis in the cases presented in this paper was found in emotionally unstable individuals subjected to severe social and environmental stress.

5. It is possible that these cases represent manifestations of the alarm reaction.

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LXXX

BILATERAL GRANULOMA OF THE LARYNX FOLLOWING INTRATRACHEAL ANESTHESIA

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The many advantages of the method of intratracheal intubation in anesthesia have, through four decades, become too well known to warrant comment. Since the successful use of intratracheal insufflation in a human being was first accomplished by Elsberg[1a] in 1909, increasing use of intratracheal anesthesia by hundreds of anesthetists in thousands of cases has made this method an invaluable aid to surgeon and patient alike. The comparatively stable position it has maintained during the years it has been in use at the Mayo Clinic is set forth in a compilation of the relative frequency of use of the various special agents and methods employed there.[1b] In 1929, the first year, the percentage of cases in which it was used was 2.9; in 1933, 16.2; and in 1938, 1939 and 1940, 13.1, 13.9 and 13.8, respectively. In 1930, with the coming into use of the Magill tube, it was employed in 300 cases; in 1940, 2,309 cases.[1c] Its greatest usefulness is for operations about the head and neck, with neurologic operations next in frequency. The method is also a very useful adjunct to inhalation anesthesia for intrathoracic operations. Many surgical procedures, particularly in thoracic surgery, would, in fact, be impossible without it.

Of the few untoward sequelae of intratracheal anesthesia, one is granuloma of the larynx. The paucity of references in textbooks and the dearth of reports in the literature indicate the rarity of occurrence of this anomaly, or at least the infrequency of its recognition in association with the anesthesia, since it appears that it may be a surprisingly late sequela. In view of the widespread use of this method over a long period of time, it would seem that the literature should yield more than the nine cases of granuloma of the larynx attributed to this cause, reported by eight authors, which were all that an exhaustive search disclosed.

REVIEW OF THE LITERATURE

Many textbooks make no reference to granuloma of the larynx following intratracheal anesthesia. Jackson and Jackson[2] mentioned that nonspecific granuloma, in contradistinction to specific granuloma arising as a manifestation of specific systemic infections such as syphilis or tuberculosis, is most commonly superimposed on a contact ulcer. In their classification of causes of laryngeal trauma, under medical and surgical accidents these authors included misdirected intubation tube.

Lundy[1d] observed that complications associated with the use of the intratracheal tube are due principally either to the trauma of introducing the tube into some part of the respiratory tract which is not patent enough to accept it without injury to the tissue, or to compression of the tube during anesthesia. He stated:

"In a rare case the surface of one or both vocal cords is traumatized and a small blister or hematoma appears, which is usually associated with hoarseness. Occasionally when this has happened the patient complains for months after operation of hoarseness and on examination a small lesion (granuloma) may be found on one or both vocal cords. In the one or two cases in which I have observed this result the lesion was treated successfully by the oral surgeon with a protected diathermy needle. That the lesion is due to the tube cannot be determined with any certainty since contact granulomas have been found in cases in which an intratracheal tube has not been used."

Flagg[3] observed that a small abrasion of the mucous membrane covering of the cords usually gives no difficulty, unless it is followed by secondary infection, and added that it is most often due to intubation with a dry tube during spasm of the cord.

British authors contributed the first three of the nine cases reported in the literature. Clausen,[4] in 1932, reported a case in which a young woman was anesthetized for the extraction of two wisdom teeth, the method used being the passage of a soft rubber tube through the nose into the larynx under ether anesthesia, continuing with nitrous oxide and oxygen through the tube. This intubation, performed with the aid of a laryngoscope, was difficult. Some time after uneventful recovery, the patient complained of hoarseness which did not respond to treatment and a feeling of something present in the larynx. Six months after administration of the anesthetic, a granuloma was found at the posterior end of the larynx, growing from the right side below the vocal cord. A few days later, she coughed up the entire tumor, which was the size of a small pea and composed entirely of blood clot showing little early organization, with the smooth surface composed of fibrin.

In the case described by Gould[5] in 1935, anesthesia was induced by nitrous oxide and ether, delivered through a nasal endotracheal

tube over a period of three hours and ten minutes. The Magill tube was introduced blindly without difficulty. The patient, subjected to hysterectomy because of carcinoma of the cervix uteri, made an uneventful recovery. Three months later she was readmitted to the hospital as an emergency following the second of two severe attacks of dyspnea and cyanosis within the week. In the interim, "loss of voice," especially while she was singing, had occurred with increasing frequency. A subglottic granuloma was removed, and two months later a slight recurrence of the growth necessitated curettement. Histologically, the specimen had the appearance of granulation tissue undergoing degeneration. There was no indication of neoplastic or tuberculous process, and the patient experienced no further trouble.

In 1938, Cohen[6] reported a case in which cholecystectomy was performed under nitrous oxide-oxygen-ether anesthesia. A medium sized Magill tube was inserted by the blind nasal route after repeated attempts, and the operation was completed in 55 minutes. Ten weeks later, the patient complained of an unstable voice and a sensation of something moving up and down in her throat, symptoms of nine to ten weeks' duration, preceded by no previous hoarseness or other throat trouble. A small tumor at the base of the right vocal cord was removed without recurrence. The pathologic diagnosis was a polypoid granuloma of unspecified nature from a vocal cord, composed of fibrous tissue and numerous capillaries lined with larger endothelial cells. It was infiltrated with lymphocytes and plasma cells, and, superficially, polynuclear neutrophils covered with fibrin.

In Smiley's case,[7] published in 1940, endotracheal cyclopropane was used for 1 hour and 35 minutes to induce anesthesia during an operation for stenosis of the common bile duct. A soft tracheal catheter was introduced under direct vision. Aside from expectoration of slightly blood-tinged mucus during the first few hours postoperatively, convalescence was normal. The patient first complained of hoarseness on the tenth postoperative day, and five days later laryngeal examination revealed a small polyp in the left side of the larynx, which 18 days later had increased in size and seemed to hang by a pedicle. The patient refused removal and six weeks later was readmitted to the hospital complaining of difficulty in breathing and inability to speak above a whisper. Indirect laryngoscopy revealed a large polypoid swelling below the left vocal cord. On removal of the soft, rounded, pedunculated mass, the microscopic diagnosis was "granulation tissue with acute and chronic inflammatory changes (pyogenic granuloma)." Another small poly-

poid mass was removed two months later, with no further recurrence.

Farrior[8] reported a case of contact ulcer of the larynx in 1942 in which surgical anesthesia was maintained with intratracheal administration of cyclopropane for 3 hours and 25 minutes, during which time a "papilliferous adenocarcinoma" involving the left anterior and middle ethmoid cells was removed. The intratracheal catheter was inserted under direct vision. The patient returned six months later complaining of intermittent hoarseness. A small proliferative mass was attached to the free border of the right vocal cord over the vocal process of the arytenoid cartilage, with slight hyperemia of the left vocal process but no visual evidence of ulceration. The exuberant granulation tissue on the right vocal process was removed and on histologic section showed chronic pyogenic granulation tissue.

The first bilateral case was reported by Kearney[9] in 1946. Under intratracheal ethylene ether anesthesia through a Magill tube, the patient was operated on for the removal of a cyst of the left upper alveolus. Nasal intubation was accomplished with moderate difficulty under direct vision with the laryngoscope, and the anesthetic was administered for 1 hour and 25 minutes. Pain in the throat postoperatively subsided in about one week, but recurred in one month accompanied by hoarseness. A granuloma was present on the posterior third of each cord in the region of the vocal process; both were definitely pedunculated 12 days later. Operation was deferred on the advice of an internist, but within two weeks became necessary as an emergency measure because of dyspnea. The pathologic diagnosis was "granulation tissue polypi of the larynx."

In 1947, Tuft and Ratner[10] added to the literature the second report of a bilateral case. Their patient was subjected to total gastrectomy for carcinoma under ethylene ether anesthesia through a Magill tube. The operation required 3 hours and 45 minutes. On a second readmission to the hospital six weeks postoperatively, she complained of hoarseness which had persisted since the operation and had increased in severity during the three weeks immediately preceding this admission until she had become practically aphonic. Two pedunculated granulomatous masses about the size of a pea, apparently attached to the vocal processes of the cords at the posterior commissure, almost completely occluded the posterior half of the larynx. Upon removal of the tumors, histopathologic study demonstrated granulation tissue with organization.

Two cases were reported by Barton,[11] also in 1947. In the first case, four months after subtotal hemithyroidectomy for colloid adenomatous goiter, the patient complained of increasing hoarseness of six weeks' duration and occasional choking episodes. Arising below the right vocal cord just anterior to the vocal process of the arytenoid cartilage, a smooth, soft, rounded, dark red mass occupied about one-half of the trachea. The pathologic report on the specimen taken for biopsy was granulation tissue showing active inflammation. On removal of the tumor, 1 x 1 x 0.8 cm in size, the pathologic diagnosis was "granuloma pyogenicum."

The second case reported by this author was that of a man whose chief complaint was coughing with occasional hemoptysis and increasing hoarseness of one month's duration. Three months previously he had been operated on for the removal of a craniopharyngioma under endotracheal anesthesia maintained for five hours. Indirect laryngoscopy revealed a smooth, grayish, polypoid mass attached to the upper surface of the posterior third of the right vocal cord and to the vocal process of the arytenoid cartilage. Pathologic diagnosis of the specimen taken for biopsy was "acutely inflamed granulation tissue." The remaining granulation tissue had not been removed at the time the report was made.

The following report of the case which came under my care is the tenth to be added to the literature. It is the third report of a case in which the granuloma occurred bilaterally.

REPORT OF A CASE

Mrs. L. G., aged 32 years, had a thyroidectomy on April 29, 1947, for thyrotoxicosis. The anesthetic for this procedure was administered by an anesthesiologist and consisted of intravenous pentothal sodium with curare and intratracheal nitrous oxide and oxygen. The intratracheal anesthesia through a Magill-Foregger tube, size 35, was continued for a period of 1 hour and 40 minutes. On preoperative examination by the anesthesiologist, the larynx was reported normal.

The intratracheal tube was introduced by the anesthesiologist under direct vision using a laryngoscope. No difficulty was encountered during the introduction of the tube. At the time the anesthesia was discontinued and on removal of the intratracheal tube, the larynx was again inspected under direct vision by the anesthesiologist. No gross trauma of the larynx or cords was noted, but a small amount of blood-tinged mucus was observed in the larynx.

Within two hours after returning to her room the patient coughed up a sizable blood-tinged mucoid plug. For four days

following the operation, she complained of severe sore throat and a distressing dry paroxysmal cough. The cough was initiated by any attempt at talking. There was, however, no hoarseness. When she was discharged from the hospital seven days after the operation, there were no symptoms referable to the throat or larynx.

Two weeks after the patient was discharged from the hospital, there developed a mild infection of the upper part of the respiratory tract. The chief symptom at this time was hoarseness. The symptom persisted and became progressively worse for a period of two and a half months although the original respiratory infection subsided within a week of its onset. Because of the hoarseness, she was referred to me on July 22nd for laryngeal examination.

Indirect laryngoscopy revealed the following: The left vocal cord was obscured from view in its posterior two-thirds by a reddish polypoid-appearing mass which seemed to be attached to the left cord, although its point of attachment could not be visualized because of its size. This mass also obscured the posterior one-third of the right vocal cord. The anterior one-third of the left vocal cord and the anterior two-thirds of the right vocal cord appeared to be entirely normal. Movements of the cords were seemingly unrestricted, and the laryngeal airway was considered adequate. There was complete aphonia. A diagnosis of laryngeal polyp was made.

During the next two months the larynx was examined at two-week intervals. The only treatment during this period was complete voice rest. A thorough examination of the chest by an internist gave negative results, as did examination of the sputum. The blood gave a negative Kahn reaction, and a complete blood count was within normal limits.

A search of the literature during this period revealed six similar cases in which the diagnosis of granuloma of the larynx was established. On the basis of this review, the clinical diagnosis was changed to granuloma of the larynx following intratracheal anesthesia. The long period of observation was intentional because previous observers had noted that such a granuloma, if given time, would become pedunculated, thus facilitating its removal at operation. This observation proved true in this case. Just prior to operation, examination of the larynx showed the mass, which was attached to the left vocal cord, to be generally somewhat smaller and attached to the cord in its posterior third by a definite pedicle. Because of better visibility the right cord could now be seen in its entirety. For the first time a very small pedunculated tumor was seen attached to the right cord just opposite that on the left cord.

The tumor mass on the right cord was approximately one-quarter as large as that on the left and could only be seen when the patient attempted phonation, for during inspiration it extended subglottically by a thin pedicle.

Operation. On September 14th the patient was admitted to the hospital with a diagnosis of granuloma of the larynx. The following day direct laryngoscopy was done with the use of a combination of 2% pontocaine hydrochloride for local anesthesia, and pentothal sodium with curare for general anesthesia and for relaxation of the larynx. The larynx was exposed with an adult-size Haslinger laryngoscope. All structures were normal in appearance except for the vocal cords. A pedunculated, gray, granular-appearing mass approximately 5-7 mm in diameter was found attached to the upper surface of the left vocal cord at the junction of its middle and posterior thirds. A similar lesion approximately 2-3 mm in diameter was found attached to the right vocal cord in a like position just opposite that on the left vocal cord. Each tumor was removed with an angular laryngeal biting forceps. Care was taken to remove no normal cord with the tumors. Following the removal of the tumors and their pedicles flush with the cords, the larynx appeared normal. No bleeding was encountered.

The day following the operation the patient was discharged from the hospital with a good voice. There has been no recurrence of laryngeal symptoms, and ten months following the operation the larynx appeared normal.

The pathologist's report on the tissue removed from the larynx follows:

Pathologic Examination. "Microscopically, this lesion has the appearance of an inflammatory granulomatous process, proceeding to healing. There is a stroma matrix, composed of areolar connective tissue and some elastic fibers. Toward one free edge of the stained section there is granulation tissue, composed of a cellular layer mingled with tissue debris, red blood cells and various leukocytes. The latter include mononuclear phagocytes in considerable number, polymorphonuclear neutrophils and eosinophils, plasma cells and lymphoid round cells. Deposits of fibrin near this same edge support capillary blood vessels, newly forming. A prominent feature here, too, is the occasionally dispersed focus of large macrophages, sharply outlined by clear cytoplasmic content and having deeply staining, rounded nuclei. Again, in a brief statement, this material gives an immediate impression of inflammatory granulation structure, and one that is of nonspecific character.

"Diagnosis: Nonspecific granulation tissue in a mucous membrane."

Of the ten cases of which there is record in the literature reviewed, including the one case described here, nine occurred in women whose ages ranged from 32 to 60 years, averaging 45 years; the remaining case occurred in a man aged 54 years. The period of administration of intratracheal anesthesia varied from 55 minutes to five hours. In all cases hoarseness, varying in degree, frequency of occurrence, time of onset and period of duration, was the one prominent symptom. Dyspnea and cyanosis made an emergency operation necessary in two instances.[5, 9]

In six cases the granuloma occurred on the right side, in three bilaterally, and in one on the left side. The favored site appears to be the posterior portion of the vocal cord or on or near the vocal process of the arytenoid cartilage where the lesion is subject to trauma in the use of the voice. In two cases the lesion recurred, necessitating removal within two months.[5, 7] Except for Barton's second case,[11] in which the operative procedure had not been completed, cure eventuated in all cases.

Gould[5] early condemned the practice of pushing the tube against a closed glottis or pulling it up and down the pharynx in an attempt to force a reluctant entrance into the trachea when the head is in the wrong position or the patient not sufficiently relaxed. He observed that the necessity for great gentleness and for sterility is not always realized.

Smiley[7] concluded that the larynx may react to trauma by the formation of an infectious granuloma such as is infrequently observed on the nasal septum. In view of the lesions arising from the region of the vocal process of each vocal cord in his bilateral case, Kearney[9] suggested that the tumor may generally originate in a traumatic ulcer of the cord exposing the superficial cartilaginous vocal process of the arytenoid; a granuloma forms and continues to grow with the ulcer meanwhile healing slowly from the periphery, producing a smaller and smaller base for the lesion with gradual formation of a pedicle. This author thought the healing process may have progressed to the point of amputation of the pedicle in Clausen's case,[4] in which the patient coughed up the pea-sized tumor about six months after anesthetization with intratracheal anesthesia.

In my case, the role of the respiratory infection occurring three weeks postoperatively is problematical. It may have been the im-

mediate precipitating factor since the sore throat and cough complained of the first few days after the operation had disappeared more than two weeks prior to the onset of the infection. On the other hand, it may have been merely contributory, or even non-contributory, to a pathologic process already advancing. Hoarseness was first experienced in association with the mild infection of the upper part of the respiratory tract and persisted from that time on.

Lundy[11] observed that there is a type of patient who cannot tolerate any trauma to the mucous membranes and cited a case in which he observed tracheitis after the use of an intratracheal tube and urethritis after catheterization in the postoperative period. He regarded the use of the intratracheal tube as probably contraindicated in some patients who have an acute infection of the upper part of the respiratory tract. This author noted an untoward result only once when the tube was left in place for 15 hours or less, but he added that when prolonged artificial respiration is carried out and the tube remains in the trachea for periods up to 70 and 80 hours, it is observed at necropsy that a membrane has been formed much like that found about a tracheotomy tube and in addition, ulcers and discolored pressure areas may be seen where the tube has pressed against the mucous membrane. He attributed complications following the use of the tube more to trauma associated with the introduction of the tube than to the tube simply lying in the trachea for the period of the operation except when proper technique is not rigidly followed.

SUMMARY

The meager literature pertaining to granuloma of the larynx following intratracheal anesthesia is reviewed.

A case is reported in which this anomaly occurred bilaterally. It is the tenth case to be added to the literature and the third in which the lesion was biltaeral.

This tumor of traumatic origin appears most likely to arise from the posterior portion of the vocal cord or in the region of the vocal process of the arytenoid cartilage. Its removal is facilitated by waiting, when expedient, until the growth has become pedunculated.

Hoarseness following the use of intratracheal anesthesia is the outstanding symptom and should suggest repeated laryngeal examinations.

Earlier and wider recognition of the possibility of the occurrence of granuloma of the larynx following intratracheal anesthesia

may obviate this sequela of an important and, in some instances, practically indispensable method of anesthesia. Gentleness during intubation may well be stressed as the watchword.

1200 KUHL AVENUE.

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LXXXI

ORAL AND PHARYNGEAL MONILIASIS

REPORT OF TEN CASES

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This is a report of ten consecutive cases of oral and pharyngeal infection with various localizations in which *Monilia albicans* was recovered either alone or predominantly among other organisms present.

Discussion of this topic is timely because the causative agent and the disease were connected for the first time over one hundred years ago, in 1847. The organism was called first *Oidium albicans*, then *Monilia albicans*. The latter term is used generally today although, according to microbiologists, *Candida albicans* should be substituted.

The circumstance that even single cases of clear cut oral infection in adults due to *Monilia albicans* were generally considered as uncommon clinical occurrences, suggested the reporting of the findings in ten patients under recent observation.

Before studying pathological conditions with the interest centered on monilia, one has to remember that Todd[1] found by culture studies that 14% of one thousand normal individuals harbored *Monilia albicans*. In 7%, the organism was present in both the mouth and the throat, in 3.1% it was obtained from the mouth only, and in 3.9% from the throat only. There was a higher incidence in females, as 18.2% of 527 females yielded the germ, while it was present in only 9.3% of 473 males.

Moreover, monilia is commonly present in the sputum obtained from patients suffering from respiratory disease.[2] It was found that demonstration of the monilia in the sputum is not evidence of its presence in the bronchi, and that bronchial moniliasis can not be diagnosed by the examination of the sputum alone. This is another

example of the fact that the presence of monilia is not equivalent to moniliasis.

The yeast will grow in the oral cavity under exceptionally favorable conditions and if circumstances favor its growth, the infection is even transmissible.[3]   Debilitating systemic disease, diabetes, and, before all, unhygienic conditions, will facilitate the establishment of moniliasis, although an entirely unexplained fact is that the previously present monilia may all of a sudden become pathogenic.[3] Characteristically, this appearance under abnormal circumstances is the condition called "denture sore throat." This is pathologically a subepithelial inflammation and not an erosion, and it is probably due to infection with a yeast of the genus *monilia* as the predominant factor.[4]   The material of which the denture is made plays a very small part and the condition is seen in its severest forms in debilitated subjects.   Antiseptic treatment of both plate and tissue brings about definite improvement, without any attention to contributory factors.   This indicates that the infection can be fought by restoring the hygienic balance but no explanation is offered for the fact that monilia present in a healthy individual may suddenly assume a pathogenic role.

The classical bibliography of Bailey Kelly Ashford[5] gave a picture of the importance of similar infections in tropical hygiene; while, as pointed out by Frost, Sutherland-Campbell and Plunkett,[6] monilia infection of any sort was considered unusual in adults in temperate climates.   Even a single instance seemed to merit reporting when oral moniliasis was associated with other lesions, as in the patient of Frost and his collaborators,[6] with invasion of the face, axilla and the groin.

Whenever monilia is found in a healthy person, the possibility must be considered that previous pathological conditions, hitherto unobserved, left the patient in the state of a carrier.   This is due to the fact that symptomatology is frequently negligible in the course of this disease.

Since M. *albicans* was isolated by Lägenbeck in 1839, its presence was recognized in soil and dust particles.   Any substance contaminated with dust is a potential source of infection.   That the infection is not more frequent, can be explained by the lack of its aggresive invasive factors.   It flares up when progressive debility obtains, as sometimes in diabetes or pregnancy.

Monilia can be present in association with other organisms. Robinson and Moss[7] considered it doubtful whether the other species

of yeast-like germs obtained were the cause of the lesions from which they were isolated. They were able to recover monilia from perlèche lesions and from a number of complicating mycotic lesions, and found that a superficial glossitis from which M. *albicans* can be obtained occurs sometimes in associated lesions, interdigital erosion, paronychia and dermatitis of the toes.

Oral and pharyngeal manifestations are not everyday findings and are less frequently encountered as infections of the female genitalia, the skin and the deeper airways.

Hofstadt and Wheelon[8] stated that there have been few reports concerning infections of the tongue due to monilia and that practically all such cases have occurred in conjunction with some other form of infection. In their case an atypical strain of *Monilia bronchialis* described by Castellani[9] was isolated from the patient's tongue. Laessle's patient[10] showed extensive oral lesions and bronchomycosis.

In nurseries, oral moniliasis is more common, and the investigations of Anderson, Sage and Spaulding[11] give information about the importance of the presence of other fungi. In their series of infants, oral thrush developed in each instance in which M. *albicans* was found in the mouth of a newborn infant, but in no instance where any other forms were present. Furthermore, M. *albicans* was isolated, without exception, from the mouth of each infant who had oral thrush. After an infant had had clinical thrush, M. *albicans* was harbored in the mouth for a variable period even after all clinical evidence of the disease has disappeared.

Intimate postpartum contact, as at nursing time, may be significant as to the introduction of M. *albicans* into the mouth of the infant from a mother with vaginal moniliasis, whose skin, clothing, and bed are contaminated.

According to Robinson and Tasker.[12] monilial infection of the mouth is common in infants and is only occasionally observed in children and adults. Skinner's analysis[13] of other statistics shows a higher incidence.

Robinson and Tasker[12] mention instances of a chronic latent type, resistant to all known therapy: reports on development of leukoplakia and epithelioma indicate that chronic latent oral moniliasis in adults should be classified as precancerous. They describe the case of a girl with chronic lesions of twelve years' duration in which the mother had both oral and vaginal infection of the same type.

The role of debilitating conditions seems to be reduced some-times to a minimum as shown by the observations of Tumulty and Michael.[14]   They described 18 patients with monilial pharyngitis, all well nourished young soldiers in good general health.   These cases of acute membranous pharyngitis were discussed under the follow-ing headings: Group 1: Monilia was the only pathogenic organism group on culture of materials from the throat and was considered to be the agent responsible for the inflammation.   Group 2: M. *albicans* and beta hemolytic streptococcus were grown but the monilia was considered to be the agent responsible for the inflammation.   Group 3: M. *albicans* and beta hemolytic streptococcus were grown in pro-fusion on culture and it was impossible to determine which agent was responsible for the inflammation.   Group 4: Monilia was grown inconstantly in small numbers in the presence of either beta hemo-lytic streptococcus or *Corynebacterium diphtheriae* and was thought to play no role in the inflammation.   Group 5: Monilia was grown in absence of acute inflammation in company with diphtheria bacilli and beta hemolytic streptococcus.   The authors emphasized that cases in which monilia was thought to be solely responsible for the production of acute pharyngitis, the latter was indistinguishable from any acute pharyngitis due to either beta hemolytic streptococ-cus or C. *diphtheriae*.

The duration of the condition varies greatly.   Pels, Dresel and Salinger's patient,[15] aged 7 years, had had thrush in his mouth since the age of 9 months.   Occurrence of an eruption on the face, neck and the fingers following the presence of lesions in the mouth, after an interval of about six years, was reported.[16]

As on mucous membranes, the presence of monilia on normal skin was described; but M. *albicans* could not be demonstrated in even a single case, as reported by Benham and Hopkins.[17]

There are several observations on M. *albicans* causing inflam-mation of the skin of the external auditory canal.[18]

MATERIAL AND METHODS

Abundant scrapings of material of all the observed lesions were planted on four different media: Sabouraud's, Loeffler's, blood agar and cooked meat broth.   After this, pure colonies were selected and slide cultures were made.

COMMENT

In general, this entire group of patients, all of whom were seen in private practice, were in good general condition.   All were living
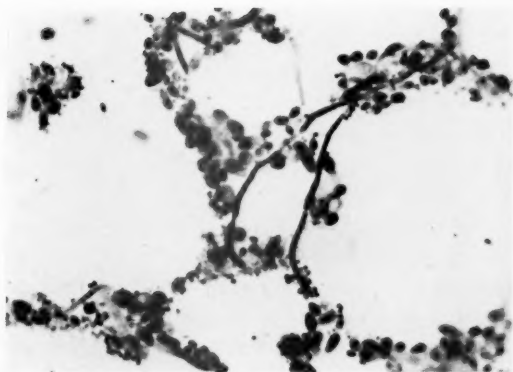
Fig. 1.—Clusters of spores around the mycelium in *Monilia albicans* (slide culture).

under comfortable social conditions. No debilitating causes figured in the history and no infections of similar nature occurred in their families.

In this group, contact with, or the presence of, pre-existing disease could be practically excluded, even more so than in the case of the 18 soldiers described by Tumulty and Michael,[14] where at least infection or contamination from environment was possible. There was no connection with any wasting disease such as diabetes. On the other hand, when obesity was present, it had no bearing on the course of the illness or on the result of the therapy. Presence or absence of dentures did not seem to play any role, possibly because the mouth had been kept under good hygienic care.

All except two of the ten patients admitted being heavy smokers but except for this high figure no reason could be seen to connect this fact with the appearance of the disease. The patients were not asked to radically change their smoking habits and so, after resolution, no further importance could be attached to this factor. Only one patient (Case 9) admitted being a chronic alcoholic. He had a so-called geographic tongue with perlèche.

M. albicans was found alone in six cases, while as associated infection, beta hemolytic streptococcus was present twice and the following once each: alpha hemolytic streptococcus, gram-positive diplococci, gram-negative rods, *Escherichia coli* and *N. catarrhalis*. None of these associated bacteria seemed to alter the clinical picture, ex-

cept in two cases where the beta hemolytic streptococcus may have been responsible for the slightly elevated temperature (Fig. 1).

Higher than normal temperature was recorded in four patients, with 100.0° F. as the highest mark. Even this slight elevation disappeared after a few days.

Half of the dates of the first visits fell into the months of June and July; but, with varying periods of duration of the symptoms previous to the first visit, no seasonal relationship was indicated. The duration of the complaints previous to the first examination was from one week to twelve months, with the data unknown in two of the cases. The patients were generally not unduly disabled by their condition. There were two exceptions to this. A generally unstable, very nervous woman (Case 7) was highly preoccupied by her condition; and a man (Case 4) was excluded from every kind of work by transitory edema of the epiglottis.

There were six male and four female patients and their ages varied between 33 and 79 years with an average of 46 years.

The symptoms from which relief was sought were sore throat, sore and dry tongue, the latter in spite of the almost constant salivation. One patient presented the paradoxical picture of complaints about a very disturbing dryness of the tongue while constantly drooling saliva.

The patient with the edema of the epiglottis (Case 4) presented the only instance in which the manifestations were outside the boundary of the oral cavity and the pharynx. This patient also showed no tendency of invasion in the direction of the glottis. There were no concomitant findings in the deeper airways, genitalia or on the skin. One patient of this group (Case 3) complained about wheezing and a productive cough, but the lungs were clear and the x-ray findings were negative.

A slight amount of edema was observed around the white patches and sometimes the patients complained that their tongues became larger, getting in their way.

The local manifestations included extensive creamy white, furry patches on different parts of the tongue and the pharynx, on the cheeks and the gums and, in one case, the epiglottis. Contrary to the generally found description, there was no bleeding when the patches were wiped away; thus, they could hardly be confused with the firmly adherent membranes of diphtheria. Moreover, their diffuse distribution was very characteristic as against the more restricted localization of diphtheria (Fig. 2).

Fig. 2.—Showing surface of tongue covered with thick coating of *Monilia albicans*.

The most conspicuous feature of these ten cases was the frequent association of "white furry" patches with "black furry" patches, also known as hairy or black tongue. In the literature, the simultaneous presence of these two conditions has been very infrequently reported. Black or hairy tongue has been described as due to *Cryptococcus linguae pilosae* in symbiosis with *Nocardia lingualis Castellani*.[3] There is no report in available literature of its being due to *M. albicans* alone.

Yet, in ten patients infected with *M. albicans*, three showed only white patches; four presented predominantly darker areas; in three others both dark and white patches were present in approximately equal extent.

Hitherto, *M. albicans* has been considered as causing only white areas. Should the observations presented here be confirmed by further studies, it may be considered that *M. albicans* is a predominant organism in black or hairy tongue patches as well.

Again, *M. albicans* has been described as the causative factor of delicate fibrinous patches. In these cases, it was demonstrated as causing furry plaques of shades from white to dark.

This is in line with Senear in his discussion of Sage's "Oral Lesions"[19] when he emphasized that monilial infections are not confined to the typical white patches.

Therapy in all cases was conducted on simple lines. Local applications of gentian violet were administered: painting with a 10% solution in alcohol 1:10,000 and as a gargle in a watery solution of 1:100,000. Large doses of vitamin B complex were given by mouth. This regime cleared up the condition even in cases in which several other medicaments such as neo-arsphenamine, perborate, and penicillin had been tried previously by others without result. The treatment had to be continued from one to ten months with an average of three and a half months. Rare recurrences were successfully attacked with the same applications.

As black hairy tongue is recognized as a tenacious disease almost irresponsive to therapy, we may have seen here merely a darker variety of the generally white monilia patches, which can be present either as delicate membranes or more elevated, even furry, plaques.

Details regarding the connection of this condition with a "true" hairy tongue remain to be elucidated. The therapy mentioned above was equally successful against patches of very different hues; a further reason to consider the manifestations reported in this paper as a clinical unit.

SUMMARY

Ten cases of oral and pharyngeal inflammation in adults are described, with *Monilia albicans* isolated as the only or the predominant organism.

There was frequent association with the condition called black or hairy tongue. On the basis of the common causative agent and the uniformly successful therapy, there may be justification to consider these furry patches of all hues between white and black, as belonging to the same clinical entity.

520 COMMONWEALTH AVENUE.

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LXXXII

THE EARLY TREATMENT OF WOUNDS OF THE FACE AND NECK

Morris Swartz, M.D.

Boston, Mass.

This paper is being presented to record the experiences and observations of an otolaryngologist in the early treatment of wounds of the face and neck in war surgery.

The unit to which I was attached was a 400-bed evacuation hospital. It was conceived with the idea of having a self-sufficient mobile unit containing all possible branches of medicine and surgery as close to the front lines as possible. Its place in the chain of evacuation of the wounded was such that patients were seen from a minimum of a few to a maximum of about 18 hours after injury and kept ten days or longer if necessary before being evacuated to the rear.

The majority of the observations were limited to a ten-day period, due to circumstances of war. Therefore the only evaluations that will be made are those that are significant in saving life within a ten-day period, such as control of hemorrhage or infection, or both.

The role of the department in this type of unit was that of a face and neck department committed to traumatic work and endoscopy. The department often worked in conjunction with the neurosurgeon, the maxillofacial and dental groups, the thoracic surgeons and the anesthetists in their fields. Only when combined specialists worked together pooling their knowledge were the best results obtained.

In the early stages of the campaign the Ear, Nose and Throat Department was relegated to a very minor role, but it soon became self-evident by sheer weight of numbers (19% of all wounds in our unit were of the face, neck and head) that the department demanded a major role in the hospital organization, and finally got it.

Wounds of the Face. Soft tissue wounds of the face varied from minor ones to gaping wounds of the entire facial structures. The pattern of early treatment in these cases was set up as follows:

Attention was given to thorough cleansing of the wounds with soap and water until all gross evidence of contamination with dirt, clothing, etc., was removed and there was fairly good free bleeding from the subcutaneous tissue. This was followed by primary closure. Approximation of the oral mucous membrane, subcutaneous tissue, and skin was done in that order. Fine black silk sutures were used in the mucous membrane, plain catgut in the subcutaneous tissues, and black silk in the skin. Sulfadiazine powder was dusted in the wound and penicillin given parenterally throughout the remainder of the hospital stay.

One will notice that primary closure was used on wounds of the face contrary to what was taught and practiced on other parts of the body in war surgery. This was done with the following premises in mind:

1. The extremely rich blood supply of the face minimizes chances for infection.

2. Penicillin on hand extends the golden period for primary wound closure from 8 to about 18 hours.

3. Unquestionably more favorable cosmetic results are obtained in primary closure of wounds of the face.

When, however, the patient was seen more than 24 hours after being wounded, and there was evidence of gross infection, no attempt at primary closure was made. This lesson was brought home forcibly by one case; that of a prisoner of war with a wide gaping wound of the face and lower jaw, who had been lying in a field for two days after being hit. Despite what I thought was adequate attention to the toilet of the wound, débridement and chemotherapy, the wound broke down entirely within 48 hours after primary closure. It had to be widely re-opened and left that way for seven days, with hot soaks before all infection was overcome enough to attempt a closure. It was an excellent example of what not to do and how much more important good judgment was than penicillin.

Since, however, this was the only case that broke down and became infected within a ten-day period, and since this patient had been greatly exposed to infection for two days, I felt justified in our initial stand of treating wounds of the face received within an 18-hour period by primary closure.

Shock—In Wounds of the Face. One of the most amazing phenomena that was called to my attention very early in the cam-

paign was the relative absence of shock associated with massive wounds of the face and lower jaw. Lesser wounds in other parts of the body were associated with a greater degree of peripheral vascular collapse than the ones above mentioned. I have seen patients with the whole floor of the mouth and lower face shot away and not in any apparent degree of shock. I cannot explain it. Others writing on war surgery have made the same observations. The importance of the absence of shock is that there was nothing to delay immediate surgical approach to the wound.

Wounds of the face in this paper are divided for descriptive purposes into the upper and lower halves. Wounds of the mandible and the associated structures are included in the lower half; wounds of the maxilla and adnexa in the upper half.

Bony Injuries of the Face—Upper Half. There were all possible types of fractures seen in the upper half of the face, from simple uncomplicated ones to those of hopeless comminution or complete loss of bony structure. When the bony structures of the face were subjected to high explosive or gun-shot missiles usually more than one bone was involved.

No two wounds of the upper face were exactly alike qualitatively or quantitatively. The treatment of each was individualized on its own merits and the over-all picture of the patient himself, but certain basic principles were followed in all. Many of the wounds contained foreign bodies. If they were accessible they were removed. If removing them meant unjustifiable surgery, then they were left in situ.

Wounds of the Maxillary Zygomatic Compound. These cases ranged from simple linear fractures to egg-shell comminution. Where there was a simple depressed fracture of the zygomatic compound with no other complications, reduction was done via the external Gillies' approach through the hair-line. Depressed fractures of the anterior wall of the maxilla were approached through the classical Caldwell-Luc route since in most of these cases it was found that loose fragments of bone were present in the maxillary sinus. The fragments were removed, blood clots evacuated and a naso-antral window made for counterdrainage.

These cases with egg-shell comminution presented the problems that many bony fragments could be retained, of how to obtain realignment of contour and what to do with the mucous membrane. We tried to make it a rule of thumb that those bony fragments that were attached to underlying periosteum and mucosa were to be left

in situ; the remainder were removed as foreign bodies. This worked well within the period of our observation since union appeared to be taking place. The problem of mobilization and retention of contour was solved by packing the antral cavity with vaselin gauze tight enough to maintain contour until there was enough fibrous union of the fragments to maintain its own support. This calculated risk had two possibilities: (1) Infection of the antral mucous membrane from prolonged packing and severe trauma, or (2) an inherently healthy mucous membrane being able to withstand this and not become a pyogenic one. The gauze packing was left in for five to seven days, then it was slowly teased out through an opening originally left in the buccal mucosa. The majority of the fractures maintained their position.

Personal communication from maxillofacial men in the rear echelons make me feel extremely conservative. They left packings in the antra for periods as long as 21 days with no ill effects on a previously healthy mucous membrane. They maintained that no chronic maxillary disease resulted from this procedure.

Open Wounds of the Nasal Dorsum. The familiar subject of fractures of the nose will not be discussed, but the point I wish to stress here is that those cases of open wounds of the nasal dorsum, in which there was quite a loss of skin either over the dorsum or over the ascending maxillary processes, much to my surprise lent themselves to good closure after adequate undermining of the skin without any undue stress. The elasticity of the skin in this region was amazing; no grafting was necessary.

Wounds Involving Frontal Sinuses. These were, in the majority of cases, a combined problem for the otolaryngologist and the neurosurgeon. In those cases in which fragments of bone were driven into the frontal lobes, the neurosurgeon took over until the frontal sinus question came up, then the ever vexing problem arose of what to do with the frontal sinus mucosa and particularly the nasofrontal duct when it was not involved. The neurosurgeon argued that the mucosa must be removed in toto down to, and including, the nasofrontal duct which they attempted to close by curettage or cautery. I could not see removing a perfectly healthy sinal mucous membrane, and irritating an apparently normal nasofrontal duct, thereby removing a tremendous barrier against infection in the future. They argued that a dura exposed to possible infections from the nasal cavity below would be liable to serious possibilities. That was one problem that was not solved to our satisfaction.

I had one case, however, of a depressed fracture of the left frontal bone from a vehicular accident where the anterior wall of the frontal sinus had been driven through the posterior wall, torn the dura and was lodged in the frontal lobe. Under local anesthesia the original wound was enlarged along the unshaven eyebrow and midfrontal region in shape of an L, the frontal sinus exposed and the fragment of bone found to be wedged into frontal bone higher up. After the wedge of bone was removed, the rent in the dura which extended to the superior longitudinal sinus was closed with No. 0000 black silk eye suture, a muscle flap placed over it and the skin closed tightly. Since this was after the war I was able to observe this patient for one month. He had a pulsating defect over the left frontal area and nothing else. During this stay he developed an acute upper respiratory infection with no untoward results.

Hemorrhage in Massive Wounds of the Upper Face. Another of the surprising features of the massive wounds of the face was the small amount of hemorrhage seen in these cases, at least in those that were brought to us. There were massive gaping wounds where half the upper face was torn away, but there seemed to be little bleeding from these areas, and the patients showed no signs of loss of large amounts of blood. Perhaps it was due to the blasting effect of the missiles that thrombosed or caused an angiospasm of these vessels, for, after débridement of the devitalized tissues was started there was enough good bleeding to warrant primary closure of these wounds.

Wounds of the Lower Half of the Face. Wounds of the lower jaw usually involved the floor of the mouth and its structures, and when the mandible was fractured as was frequently the case, the soft tissue work was performed by the laryngologist and then the dental surgeon would take over with his specialized knowledge of reduction and mobilization of the fractured mandible. Many of these wounds showed great swelling of the floor of the mouth with associated blocking of the airway that was alarming, so that one's first thought was toward tracheotomy to prevent suffocation. Early in our experience we discovered, through chance, that after control of hemorrhage from the vessels in the submaxillary region, within a very short time before we were through closing the wound, the tremendous edema of the floor of the mouth would vanish as though by magic, and there was an adequate airway. In these cases preliminary tracheotomy was no longer performed but first attention was given to control of hemorrhage from these vessels, and I strongly felt that doing a tracheotomy was an added unnecessary burden to an already strained body economy.

Compound fractures due to wounds involving the region of the condyle of the mandible frequently involved the internal maxillary artery which runs medial to the ascending ramus of the mandible and is very difficult to approach for control of hemorrhage. In these cases it had to be controlled by ligature to the external carotid artery.

Anesthesia. Local anesthesia consisting of infiltration of 2% novocain with a preliminary dose of 1/6 grain morphine sulfate plus 1/150 grain atropine given intravenously was the method of choice. This was first started (as are so many other procedures) due to the circumstances of necessity. Because of the dearth of anesthetists, they had to be used sparingly. Whenever possible, and at times in situations which even seemed impossible, local anesthesia was used about the face and neck, and intravenous morphia was found to be a very satisfactory premedication agent. It was used only in those cases in which there was no evidence of shock since there was the danger of pooling of the drug from previous doses administered along the lines of evacuation. It was a quick acting method that would immediately lull the patient into good sedation. No untoward effects were noticed from its administration in several thousand cases in our unit.

It was also observed that a relatively small amount of local anesthesia had to be used in these cases of massive wounds of the face, and in many instances it was first appreciated that a great deal of handling and cleansing of the wounds could be done on patients not in shock and before the local anesthesia was used. Perhaps the trauma caused anesthesia of the sensory nerve endings of the nerves involved.

The advantages of local over general anesthesia in these cases were:

1. Diminished bleeding.
2. Ease and rapidity of administration.
3. Abolishing the necessity of a long general anesthesia.
4. Prevention of blood aspiration.

Wounds Involving the Nasopharynx. These wounds were always associated with neighboring structures and served either as a resting place or a passageway for the missile. The region of the angle of the jaw was usually the point of entrance or exit. In a perforating wound of the nasopharynx this entity created no particular problem except that of inspection or palpation of the area to try to ascertain the extent of the damage; treatment consisted in

leaving it alone. The problem arose in penetrating wounds with lodgment of the missile somewhere in the walls of the nasopharynx. We were not in a position to obtain exact localization of the foreign body by x-ray examination and one is limited in direct visualization and probing for a foreign body as is done elsewhere in the body. One solution of this problem is illustrated in the following case:

CASE 3.—A young, white male soldier was admitted late one night with a gunshot wound of the left mandible just below the zygomatic arch. There was very little bleeding present. X-ray films showed a fracture of the left mandible and a bullet lodged somewhere in the region behind the right maxilla. The missile could not be palpated from the outside and its point of entrance into the right nasopharynx could be palpated as a thin slit, since the tissues had partially closed over it. Attempt at removal late that night by blind palpation through the nasopharynx was unsuccessful. We decided to try again in the morning under a general anesthesia with the possibility of an external approach. The next morning under endotracheal anesthesia another attempt was made with palpation by myself and another laryngologist, each of us working for about 20 minutes; we were unsuccessful. We then decided to split the soft palate to see if we could get better vision. The soft palate lateral to the uvula was split all the way up to the hard palate, retracted, and, to our surprise, the wound of entrance could be seen in the area above the Rosenmüller's fossa. A Kelly forceps was inserted and the bullet was easily felt and extracted. The soft palate was then sutured in two layers with black silk. At the end of about five days the sutures were removed and the wound healed completely with no interference of the normal function of the soft palate. The jaw was immobilized and the patient made an uneventful recovery in six weeks. The point I wish to stress in this case is that splitting of the soft palate gave the best possible exposure of the nasopharynx to direct vision without any apparent untoward after-effects.

Wounds of the Neck. About 2% of all our surgical admissions were wounds of the neck. They were classified by me as superficial and deep. In the anterior neck those external to the level of the sternomastoid were superficial and of no serious significance; those internal were classified as deep and serious. Most of the wounds of the posterior neck were not of a serious nature unless there was involvement of the cervical spine.

Superficial Wounds. After débridement the superficial wounds of the neck were dusted with sulfa powder and then closed tightly.

Penicillin was given parenterally. No more need be said about these wounds; no infection followed.

Wounds of the anterior portion of the neck may or may not involve the underlying laryngeal structures. When they do not, then very little débridement is done since there is very little subcutaneous tissue present external to the laryngeal structures, and those must be preserved. The wounds were closed tightly with sulfa powder and the patients put on penicillin. When there was involvement of the underlying structures, the first step was immediate low tracheotomy. The patients who survived wounds of the larynx must have been very few, since in our unit I saw only 21 cases out of approximately 14,000 surgical admissions. I am sure there were many more, but those did not survive. Open wounds of the larynx were closed tightly after tracheotomy. In cases of nonperforating wounds, tracheotomy was performed since there was enough hemorrhage and edema of the false cords and arytenoids to severely impede respiration; it was the only method to put the larynx at rest and recovery was much faster.

I had one noncombatant fracture of the larynx due to a truck accident. The patient showed the following picture: dyspnea, expectoration of blood, the head fixed in a semiflexed position on the chest, crepitus of the hyoid bone and thyroid cartilage elicited on palpation, and subcutaneous emphysema in the tissues of the left neck. Immediate low tracheotomy was done and the patient placed on large doses of penicillin. The larynx could not be inspected for three days because of the pain on extension of the neck. Direct inspection of the larynx on the fourth day showed marked submucosal hemorrhage of the arytenoids and left aryepiglottic fold, and a large rent in the left laryngopharynx which had a slough over it. The patient showed increasing subcutaneous emphysema and was treated by penicillin and watchful waiting. On the eighth day, all signs of emphysema disappeared. The patient was taking fluid by mouth, the temperature was normal and he was then transported to another unit. I did not observe shock with these wounds and injuries of the larynx, as has been recently described by others.

Wounds of the trachea usually were complicated by severe emphysema of the deep structures of the neck. These were treated by tracheotomy and by splitting the ribbon muscles, and often the sternomastoids as well, followed by dissection into the deep fascial planes. The emphysema would disappear after about 24 hours, then the split muscles were closed using No. 1 chromic cat gut.

Wounds of the Cervical Mediastinum. Wide incision and drainage was the most important phase in treatment. The incisions

were either the classical ones along the anterior border of the sterno-mastoid or the collar type. The latter gave just as much room and resulted in a better cosmetic closure with less scarring. Our first case early in the campaign was a pin-point wound of the right neck at the level of the cricoid cartilage which x-ray examination revealed had not passed the midline. Wide exploration was not used; the patient was treated only with chemotherapy. On the fourth day he developed symptoms of mediastinitis, and on the sixth day he died. Autopsy showed a pin-point perforation of the esophagus with a very diffuse mediastinopericarditis and bilateral pleural effusion. Following this, endoscopy was performed in every wound of the neck to see if there was involvement of the larynx, trachea or esophagus. A probe was directed into the tract from the outside and observed under direct inspection to see if it entered any of the above structures. Wide incision, exposure and drainage down to the level of the cervical esophagus was used and, in addition, chemotherapy. There were, following the first case, 12 cases of wounds of the cervical mediastinum; of these, 9 included the cervical esophagus. In my limited experience it was found that for tiny perforations of the esophagus suture was not necessary; the perforations closed by themselves. Passage of a rubber feeding tube into the stomach was not attempted for fear of further damage to the esophageal wall. The patients were fed by vein for four days, and on the fifth, fluids were started by mouth, drains were shortened and finally removed on the seventh or eighth day. All the patients recovered under this regime.

Wounds of the Large Vessels of the Neck. The wounds that we saw were usually those made by small bits of shrapnell where the entrance was small and the skin would close over. This resulted in slow bleeding under the skin, with tremendous hematoma formations; when exposed there would be an alarming hemorrhage. There were three cases of wounds where the vessels could not be reached, identified or ligated, since they were low in the neck and there was danger of exsanguination on the table. These wounds were packed tightly and the skin sutured over the pack. On the fifth day the temperature could no longer be controlled by chemotherapy. The wounds were re-opened, the packing removed and no more bleeding was observed. All these made an uneventful recovery, and within ten days no complications followed. One common carotid that was opened by a tiny missile was sutured using No. 0000 black silk eye suture. No untoward results were observed for ten days. In some cases it was possible to suture the large tributaries to the internal jugular vein with No. 000000 black silk eye suture.

Endoscopy. Bronchoscopy was a particularly valuable aid in chest wounds, postoperative atelectasis, pentothal anesthesia accidents and also to the neurosurgeon in those cases in which there was a temporary loss of swallowing reflexes. In the latter it prevented the patients from drowning to death from their own secretions.

SUMMARY

An otolaryngologist's personal experiences with traumatic wounds of the face and neck and his observations of these for a ten-day period have been outlined. No attempt is made at scientific conclusions, or originality. Trial and error was our only method of learning to save life under the adverse circumstances of war.

BOSTON CITY HOSPITAL.

Society Proceedings

Factors Influencing the Growth of Lymphoid Tissue

ABRAHAM WHITE, PH.D. (By Invitation)

(This paper appeared in full on page 523 of the June 1949 issue.)

DISCUSSION

DR. ARTHUR W. PROETZ inquired whether substances related to epinephrine, such as ephedrine and related compounds so freely employed in nose and throat practice, have any effect on the involution of lymph tissue.

DR. HENRY M. GOODYEAR asked whether iodine has any real influence on hypertrophic lymphoid tissue. This is important in view of the widespread use of iodine in vicinities where large amounts of chlorine are used in the water supply.

DR. WHITE, in closing, stated that small amounts of almost anything they have given seem to augment the pituitary cortical secretion, but it so happens that ephedrine and iodine are two of the substances they have not used experimentally. However, minute amounts of arsenic or benzene have induced involution. The influence of benzene has been known for years. It is reasonable to assume that ephedrine and iodine act in the same way, although there is no experimental evidence to support this assumption. Anesthetics such as nembutal in minute doses also act similarly. Also it has been observed that when an animal is under nembutal anesthesia, if

adrenocortical extract is given the animal begins to come out of the anesthesia.

Radium and the Lymphoid Tissue of the Nasopharynx and Pharynx: A New Universal Applicator

W. WALLACE MORRISON, M.D.

NEW YORK, N. Y.

Dr. Morrison demonstrated a flexible applicator for the application of radium to the lateral pharyngeal areas when introduced through the nose. This increases the area which can be reached in direct application. The indications for irradiation in the nasopharynx are recurring aero-otitis, recurring acute catarrhal otitis, prevention of secondary bacterial infection and chronic suppurative otitis media, and for the reduction of hypertrophied lateral bands following tonsillectomy. Contra-indications are nerve deafness, otosclerosis, atrophic rhinitis and pharyngitis, congenital bursae, recent acute infection and large masses of adenoids. Danger to the operator should not be minimized. The maximum daily tolerance dose for the operator or nurse is 1/10 r per day. Adequate protection and safeguards should always be provided. Danger to the patient may arise from overdosage. Also the possibility of a late atrophy must be considered.

Contact Ulcer Granuloma

GORDON B. NEW, M.D.

ROCHESTER, MINN.

(This paper appeared in full on page 548 of the June 1949 issue.)

Lethal Granuloma of the Nose, Pharynx and Larynx

HENRY L. WILLIAMS, M.D.

ROCHESTER, MINN.

(To be published.)

DISCUSSION OF ABOVE TWO PAPERS

DR. FREDERICK H. HILL, commenting on Dr. New's paper, wondered whether recurrence and the necessity for re-operation were not dependent upon the type of granuloma, that is, whether completely sessile or pedunculated. In one case which was bilateral, re-operation was necessary on the side where the lesion was sessile.

DR. PAUL H. HOLINGER stated that in his experience the soft granulomas responded more readily to therapy than the fibrous types. Also he has noted that pain is apparently not always referred to the site of the granuloma, being frequently experienced in the opposite cord. Voice training is very important for these patients following surgical treatment. Dr. Holinger showed several colored slides typifying the lesions under discussion.

DR. NORTON CANFIELD described two cases he had seen, similar to the ones described by Dr. Williams. Large doses of penicillin were unavailing in the last patient, who lived only six weeks after the diagnosis was made.

DR. FLETCHER D. WOODWARD cited a case in which a broad sessile mass occupied the entire left vocal cord. The lesion was removed endoscopically on four occasions, but still persisted. There was a suspicion that the underlying cartilage may have been involved and Dr. Woodward felt that it may yet be necessary to remove the arytenoid.

DR. EDWARD D. KING asked Dr. Williams whether he had obtained any results from his therapy, not having been able to finish his paper within the allotted time.

DR. NEW, in closing, stated that in his experience most of the cases of indeterminate type were sessile. Dr. Holinger's colored pictures showed the lesions most beautifully.

Regarding the soft granulomas, many of which were post-anesthetic cases, the prognosis was good, since there was recovery in all, whether fibrosed or purely inflammatory.

Commenting on Dr. Woodward's case, Dr. New has on several occasions been compelled to remove much of the arytenoid in order to get rid of all of the diseased tissue.

DR. WILLIAMS, in closing, said he had not had the opportunity of trying out the suggested therapy in a case of lethal granuloma, but referred to its possibilities because of some success it has achieved in cases of lupus erythematosus and rheumatic fever.

An Internist Looks at Specialism

JOSEPH C. DOANE, M.D. (By Invitation)

Dr. Doane reviewed the increasing trend toward specialism, pointing out the dangers as well as the advantages. Responsibility rests heavily in the very beginning on the deans and admitting committees of medical schools, in the selection of proper candidates, and the process of "cramming" so prevalent nowadays has a bad influence on the development of the future specialist. Lack of uniformity in requirements, procedures, fees for initial and re-examination among the various specialty boards, is a matter that requires co-ordinated study and action. Also there is a great tendency among specialists to refer symptoms to their own particular field. A broader base of general knowledge is essential in the making of a well balanced specialist, and there is need for better group study and co-ordination of all factors in many cases referred to specialists. Dr. Doane aptly quoted Mosher, who once said, "The strength of specialism is specialism: paradoxically its weakness is specialism."

DISCUSSION

DR. BURT R. SHURLY, after 50 years in the practice of medicine, still finds the greatest joy in knowing the patient as an individual. To be successful one must be primarily a doctor, rather than a specialist.

DR. PERRY M. GOLDSMITH, having practiced general medicine before becoming a specialist, agreed most emphatically with the speaker on the matter of broad general knowledge. Moreover, he is convinced that many specialists spend too little time in taking the patient's history. Since 60% of the patients coming to the doctor are impelled by fear or anxiety, and usually have nothing the matter with them, it is extremely important that the physician be qualified to evaluate the symptoms and set the patient aright. Overtreatment and coddling of patients with minor symptoms is to be deplored.

DR. DOANE, in closing, cited several examples of needless surgery in cases when the complete history was not properly evaluated. Also he pointed out mistakes resulting from lack of co-operation between the dentist and the internist, and the frequently needless sacrifice of teeth.

· Changes in the Nasal Mucosa Following Laryngectomy

Fred W. Dixon, M.D.

Normand L. Hoerr, M.D. (By Invitation)

AND

Julius W. McCall, M.D. (By Invitation)

Cleveland, Ohio

(This paper appeared in full on page 535 of the June 1949 issue.)

DISCUSSION

Dr. Julius W. McCall commented on the infrequency of colds in these patients. He attributes this to the fact that the absence of air currents through the nose prevents drying of the secretions and slowing of the ciliary movement which in nasal respiration frequently provide the portal of entry for bacteria. Laryngectomy patients all have moist nasal membranes and patients previously suffering from sinus infections are found to be relieved.

Dr. Harold I. Lillie corroborated the previous speaker's statement regarding moisture of the nasal mucosa. In one case where nasal crusting had been present prior to the laryngectomy, the crusting disappeared entirely without any treatment.

Dr. Arthur W. Proetz, having examined the histologic specimens prepared by Dr. Dixon, was impressed with the normal appearance of the mucous membrane taken from patients up to 70 years of age. This observation goes a long way toward substantiating a feeling he had had for a long time, namely, that inflammatory processes involving these tissues, however prolonged, may still be reversible, given ideal conditions such as prevail after laryngectomy.

Dr. Andrew A. Eggston emphasized the importance of collaboration between the clinician and the pathologist. Since the mucous membrane varies with the site, it is extremely important that the exact location from which the specimen was removed be noted. He was very much interested in the observations made in these cases and rather astounded at the minimal changes in the mucosa in the absence of nasal respiration.

Referring to the recognition of allergy in the mucosa, Dr. Eggston pointed out that plasma cells are as plentiful as eosinophils, both resulting from a hyperglobinemia.

DR. DIXON, in closing, stated that all of his biopsy specimens were taken from the anterior tip of the inferior turbinate. This was preferred because of its accessibility and because there was less bleeding and more prompt healing than had they been taken from the septum.

Bony Stenosis of the Larynx: Report of a Case

NORTON CANFIELD, M.D.

NEW HAVEN, CONN.

(This paper appeared in full on page 559 of the June 1949 issue.)

Stenosis of the Larynx

JAMES M. ROBB, M.D.

DETROIT, MICH.

(This paper appeared in full on page 566 of the June 1949 issue.)

DISCUSSION OF ABOVE TWO PAPERS

DR. BURT SHURLY pointed with satisfaction to the fine record at the Harper Hospital in the handling of these diphtheria cases. He himself had done over 500 intubations in the old days, and had seen only 12 cases in which the tube had to be retained for any length of time. Stenosis was prevented by reduction in the size of the tubes, which were often coated with aluminum or silver salts. His experience with tracheotomy in tuberculosis cases coincides with that of the speaker; namely, that the results were not good. Fortunately today with the use of streptomycin, the necessity for this procedure comes but rarely.

DR. PAUL H. HOLINGER, discussing Dr. Canfield's case, asked whether x-ray films revealed the extent and degree of the ossification. Keloid formation, more marked in certain individuals than others, may account for a greater tendency to stenosis. As for indwelling esophageal tubes, Dr. Holinger believes it is not so much a question of how long the tube is in situ as it is the degree of pressure over one restricted area. Frequent examination of the larynx is necessary, especially if there is pain and, if the tube cannot be

removed it should at least be shifted from one pyriform fossa to the opposite side.

Dr. Robb's report was indeed remarkable in that there was only one case of stenosis in a thousand intubations. One important factor in this series was the fact that when it was found that a child could not retain a tube and required a larger one than indicated for the particular age, a tracheotomy was performed in preferance. The non-cough-up tube is not safe; more cases of stenosis have been seen following its use than from the conventional tube. Dr. Holinger showed several slides illustrating various types of stenosis, ulceration and web formation.

Dr. Harold G. Tobey stated that ulceration due to indwelling duodenal tubes such as the Miller-Abbott could hardly be due to the caliber of the tube, which is very small. He explained the damage as due to the friction caused by tugging on the tube, the result of peristaltic action in the small intestines.

Dr. Canfield, in closing, stated that x-ray pictures were not taken because there was no suspicion of new bone formation. At operation this was found in the airway between the cricoid and the vocal cords themselves. The rest of the larynx looked normal, as did the cricoid itself. As to esophageal tubes, he believes the damage they cause is due to contraction of the cricopharyngeus. A new tube recently employed is collapsible. This has the additional advantage of preventing regurgitation.

Dr. Robb concluded with the comment that there is still room for improvement in the types of tubes introduced. Also, his experience during the last war impressed him with the importance of the laryngologist familiarizing himself with the procedure of endotracheal anesthesia, and making frequent examinations following such a procedure, as well as in cases where esophageal feeding tubes are in use.

Contributions of Older Fellows

As a result of a poll among the members of the Association, the following were named as having made the most outstanding contributions to laryngology:

Dr. Harris P. Mosher: Teaching and research.

Dr. Thomas H. Halstead: Early work in endoscopy.

Dr. George B. Wood: Anatomy, histology and histopathology of the tonsil.

Dr. Chevalier Jackson: Endoscopy.

Dr. Burt R. Shurly: Nutrition and diet in relation to oto-laryngology.

Dr. George M. Coates: Postgraduate education.

Dr. Fielding O. Lewis: Surgery—Malignancy of the larynx.

Dr. Harry A. Barnes: Work on the tonsils and malignancy of the sinuses.

Dr. Ralph Butler: Postgraduate teaching.

Dr. Joseph L. Goodale: Allergy—Anaphylaxis.

Dr. Joseph B. Greene: Tuberculosis of larynx.

Dr. Francis R. Packard: Medical history.

A number of these gentlemen were unable to be present at the meeting and sent letters which were read by the Secretary, in which they outlined the steps taken in their early days toward devloping the special interests through which they became so well-known to the profession. Drs. Mosher, Halstead, Shurly and Coates present-ed their outlines in person. This was followed by an evaluation and appreciation of the above contributions by Drs. Arthur Proetz and Harold I. Lillie. Dr. George M. Coates, who was Secretary for a number of years before being elected President, then presented an interesting review of many sessions of the Association during the period of his service, in which he recalled numerous outstanding programs of the day and the authors whose research and originality enriched the proceedings of the Association.

Presentation of New Instruments

Dr. John G. McLaurin, having heard Dr. Morrison's de-scription of a new radium applicator for the pharynx, was reminded of an instrument, now obsolete, which he used with success in the prevention of closure of a sphenoid aperture after operation on this sinus. He found that usually the closure was due to an excess of lymphoid tissue which yielded nicely to irradiation by means of the old-time radium needle threaded into a simple cannula left in situ for 20-25 minutes at intervals of a week.

Dr. Edwin N. Broyles presented a new laryngoscope, the proximal end of which is smaller than the distal end; this is a decided advantage when prominent teeth or a thick neck interfere with a wide exposure. To increase the vision and afford a sharper view of the details of the larynx, Dr. Broyles has devised a small proximal magnifying glass which can easily be shifted over the tube opening or moved out of the way as required.

Dr. Gabriel Tucker presented a twin-lighted laryngoscope with an attachment for oxygen insufflation, especially useful in the examination of the larynx and upper esophagus in infants. Also Dr. Tucker showed a new photographic outfit made by the American Cystoscope Company, with which it is possible to obtain good pictures of the subglottic area.

Recent Advances in Our Knowledge of Cancer

Joseph C. Aub, M.D. (By Invitation)

Boston, Mass.

Dr. Aub spoke first on diagnosis, calling attention to the most important contribution in recent years, namely, the Papanicolaou stain, which has proven of value in all localities where secretion from the region of the tumor is available. The test is significant because it depends on the known disposition of malignant tumors to slough off apparently viable cells which are readily distinguishable from cells derived from normal mucosa, which are usually old cells and not always viable. Coleman showed conclusively that neoplastic cells lack the cohesiveness which characterizes normal tissue. This accounts for the danger of implantation during surgery in malignant neoplasms.

The second important advance has been in connection with radio-active isotopes. Thus far they have not been of much help in laryngology, but in the case of the pituitary and the thyroid, radio-active iodine has already been of great service. Also radio-active phosphorus has been useful in the diagnosis of tumors of the brain.

In the nasopharynx there has as yet been very little progress insofar as the use of either of the above procedures is concerned for diagnosis.

As for therapy, at the present time surgery and irradiation are still the best we have to offer. Efforts to develop specific destruc-

tive agents against tumor cells have not been entirely successful, although the nitrogen mustards have been proven of value in lymphoid tissue and in some tumors of the lung. Since they act particularly on the actively dividing cells, they not only destroy the tumor cells but at the same time do away with a large number of the white cells in the blood stream, as well as the platelets, which may reduce the percentages to a dangerously low level. In some cases transfusions have carried the patient through the crisis to a successful cure.

Another new chemical having a selective action on actively dividing cells is urethane, which has been of some value in cases of myelogenous leukemia. On the other hand, folic acid has been disappointing in the author's experience, perhaps because his work has been in adults, whereas the favorable results reported have been mostly in children.

Dr. Aub further discussed the propensity of tumor cells to divide more rapidly than normal cells, and the problem of discovering ways and means to halt this rapid proliferation. A hint as to possibilities in this direction is the fact that carcinoma of the prostate is definitely slowed down by the use of estrogens and orchidectomy. The influence of hormones is being widely explored with varying success, but always offering a possibility of eventual solution.

DISCUSSION

Dr. LEWIS F. MORRISON mentioned the use of radio-active isotopes in malignancy of the breast. Also he has had some success in picking up malignancy of the nasopharynx by way of the Papanicolaou stain, 12 such cases having come under his observation. This was possible because of studies over a period of time relating to the normal cells recoverable in the secretions of the pharynx, whereby differentiation from tumor cells was more easily determined.

Another point that has come to the fore in this work is the fact that when biopsy specimens are rubbed over a slide and stained with the Papanicolaou stain, frequently a positive report can be obtained.

Dr. NORTON CANFIELD asked whether, in view of the tendency of malignant tumors to desquamate, one should change our present method of obtaining tissue for biopsies.

Dr. ANDREW A. EGGSTON was impressed with the tendency of certain individuals to develop malignancy or recurrences after removal which, in view of the influences of hormones already mentioned, seems to suggest a physiologic imbalance.

He mentioned the recent increase in malignancy of the sinuses as observed in his hospital. These, with the tumors of the pharynx, present a wide variety of types, many of which are highly malignant, metastasize early and, while responding early to irradiation, are prone to recurrence.

While applauding the Papanicolaou stain as a remarkable advance, Dr. Eggston feels that the clinician should not rely on it completely, since it places too great a responsibility on the pathologist. A new procedure which at times has been of value, is to rub a piece of gelfoam over the suspected area, then drop it into formalin, section and stain as for a biopsy. Frequently bits of tumor tissue are found which present typical characteristics of malignancy.

Dr. Louis H. Clerf has found the Papanicolaou stain of value in the diagnosis of endobronchial lesions, and suggests the possibility of its being used in suspected malignancy of the sinus where the secretions may contain numbers of malignant cells.

Dr. Aub, in closing, expressed a fear of biopsies in many cases because of the possibility of spreading and implanting tumor cells. Certainly the procedure should be carried out with extreme care.

He mentioned the virus question which was brought up by one of the discussors. While its effect has been proven in animal experiments, its relationship to carcinoma in the human being is still conjectural. It seems logical, however, to assume that viruses may stimulate the mechanism whereby cells are activated to rapid propagation. Also the relationship of nucleoprotein to tumor cells is very intriguing because it has been shown that cells seem to derive the energy for propagation from this biological substance. This has been shown in experiments on the liver, where amino acids can activate a small rest of tissue to over-function after the greater part of the organ has been removed. Tumor cells have shown a greater aptitude in this direction, which again brings up the great question as to why normal cells will in certain individuals tend the same way.

Plastic Repair of Saddle Nose

Edward D. King, M.D.

Los Angeles, Calif.

(This paper appeared in full on page 577 of the June 1949 issue.)

DISCUSSION

Dr. Samuel Salinger finds that the greatest objection to bone implants is the fact that the resulting nose is too rigid and incapable

of the mobility which is normally inherent by reason of the exposed position of the organ. His own preference is for autogenous cartilage and when this is not available, for cadaver cartilage of proper consistency. He recalled his experiences with all of these materials and mentioned the fact that in some instances neither cartilage nor bone will suffice. This is particularly the case in posttraumatic and postinfection cases where there is extreme retraction of the tip of the nose and columella due to dense scarring. For this type of case he has had good results from ivory, properly prepared and made up in two interlocking pieces to fill the nasal dorsum and support the tip and columella. Several cases of this type have been under observation for over 20 years with apparently no deleterious effects.

DR. FRED Z. HAVENS also commented on the rigidity resulting from the use of bone grafts. His own preference is for autogenous rib cartilage which is put into a double boiler before use in order to prevent curling.

DR. KING, in closing, admitted that the nose does feel rigid after a bone implant, but as long as it looks well and patients are satisfied, the means is justified.

Hemilaryngectomy: A Method of Maintaining a Permanent and Ample Airway

HENRY M. GOODYEAR, M.D.

CINCINNATI, OHIO

(This paper appeared in full on page 581 of the June 1949 issue.)

DISCUSSION

DR. LOUIS H. CLERF expressed interest in the procedure although he had never seen it done. Certainly it seems a pity to sacrifice half of a larynx if it could be saved.

DR. GORDON B. NEW inquired as to the grade of tumor involved. He would consider the procedure risky if the tumor were a highly malignant one, because the manipulation involved would very likely tend to scatter tumor cells into raw areas. Personally he would be inclined to destroy the tumor with surgical diathermy, which he considers safer than the cold knife. As to resultant contraction, he had had good results from the implantation of a skin

graft, having observed several cases up to three years with the graft plainly visible.

Dr. Goodyear, in closing, admitted that the procedure was new and had not been under observation in a sufficient number of cases or for a sufficiently long period to afford a complete evaluation. However, he feels that the procedure should be explored because of the possibility of rehabilitation in a certain group of cases which lies midway between the ideal laryngofissure type and the group of patients needing a laryngectomy. He agrees with Dr. New on the value of electrocoagulation, which he uses in part of the operation. As for the skin graft in cases of stenosis, he has seen the skin ultimately replaced with mucous membrane, which is contrary to Dr. New's experience. This may have been an exceptional case.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, January 3, 1949

THE PRESIDENT, DR. WILLIAM A. SMILEY, IN THE CHAIR

The Recognition and Management of Malignant Tumors of the Larynx

FREDERICK A. FIGI, M.D.

(Abstract)

Diagnostic and therapeutic procedures cannot be applied routinely in cases of malignant tumors of the larynx. Many malignant lesions in this situation conform to a definite pattern and are readily recognizable clinically. Others present special diagnostic and therapeutic problems which test the acumen of the laryngologist. Perhaps in no other specialized field of medicine does the experience of the consultant and surgeon have a greater bearing on the prognosis.

The frequency and the seriousness of malignant tumors of the larynx are now generally appreciated. It is realized also that early recognition of these lesions is necessary if treatment is to be most effective. While a number of extensive neoplasms of the larynx are controlled by therapy, as a rule the prognosis becomes progressively less favorable as the process advances.

The great majority of malignant tumors of the larynx are seen in men between 40 and 70 years of age. However, such growths may occur during the third and the second decades of life, and rarely even during the first decade. Most patients less than 10 years of age who have a malignant lesion have previously received repeated irradiation for multiple papillomas. Recognition of the fact that malignant laryngeal neoplasms may occur early in life is important from the standpoint of diagnosis and treatment. Overlooking this fact may lead to the loss of valuable time in establishing a diagnosis and may necessitate a radical rather than a conservative procedure for removal of the neoplasm.

A careful history should be taken of all patients with symptoms referable to the larynx. In addition, roentgenologic studies of the thorax, blood count, urinalysis, serologic tests for syphilis and sputum examination, if indicated, should be made and a general physical evaluation done. The larynx should be studied by indirect laryngoscopy and, if indicated, roentgenograms and tomograms should be made.

When indirect laryngeal examination reveals a suspicious lesion, further investigation by means of direct and preferably suspension laryngoscopy should be made. If indicated, biopsy should be performed. Frequently the diagnosis will be obvious from the findings on indirect laryngoscopy. However, if the lesion has been traumatized recently, is acutely inflamed or considerable edema is present, clinical recognition may not be possible.

Laryngeal examination alone will not permit differentiation between many malignant tumors and a number of benign neoplasms and granulomas. Superficial papillary epitheliomas may strikingly simulate benign papillomas; also superficial infiltrating carcinomas that have not yet interfered with the mobility of the vocal cords may strongly suggest a benign tumor or an inflammatory process. Distinguishing clinically between papillary leukoplakia and epithelioma often is impossible.

In view of the seriousness of malignant tumors of the larynx, the importance of their early recognition and the impossibility of distinguishing between such lesions and certain benign neoplasms and inflammatory conditions by inspection alone, biopsy is essential in all cases. Often this can be done satisfactorily as an office procedure using local anesthesia and indirect laryngoscopy. However, in recent years my colleagues and I have become convinced that removal of tissue for microscopic study is better done with suspension laryngoscopy.

Fresh frozen microscopic sections are distinctly advantageous in the examination of malignant tumors of the larynx. By this means a competent pathologist can give the surgeon a definite histologic diagnosis, usually in a few minutes; if the tissue is unsatisfactory from the pathologist's standpoint, another specimen can be secured without delay. Moreover, in a carefully selected group of cases it is possible to remove such tumors by suspension laryngoscopy, and if indicated this procedure can be carried out immediately.

In treating malignant tumors of the larynx complete eradication of the disease must always be the primary objective. Preservation of laryngeal function is of secondary importance but is essential if

it will not lessen the likelihood of cure. Although the most extensive surgical procedures possible are frequently required in order to remove completely cancer in this situation, unnecessarily radical removal of laryngeal growths results in needless sacrifice of the voice and of the integrity of the respiratory tract. An open operation is the generally accepted procedure for removal of carcinoma of the larynx and is absolutely necessary in the majority of instances. However, in selected cases less radical external surgical procedures or even endoscopic measures will permit removal of these neoplasms without sacrifice of function and afford an equally good prognosis.

A group of cases of malignant tumors of the larynx will be reviewed. Most of these present unusual features.

DISCUSSION

DR. PAUL H. HOLINGER: Certainly we are all pleased to have Dr. Figi give this subject in such an instructive way and I think the Society is to be congratulated. Dr. Figi mentioned the importance of a biopsy in selecting therapy as well as in establishing the diagnosis of carcinoma because of the manner in which papilloma, hyperkeratosis and other lesions simulate carcinoma. Inaccuracy of diagnosis not only influences the choice of treatment in the case, but introduces inaccuracies in statistics of cure rates by different therapeutic measures. Dr. Figi stressed, too, the value of the surgeon doing his own biopsy. I definitely concur, and all who have seen the larynx following biopsy realize the changes that can take place; the extent of the lesion cannot be demonstrated as well after as before biopsy.

We have not been able to duplicate the work of intralaryngeal surgery Dr. Figi discussed, and we envy him the wonderful results he has been able to achieve by this technique. However, laryngeal surgery is leaning more toward radical rather than conservative techniques and the intralaryngeal approach seems to bring us back to conservative surgery. Our recurrences have occurred in those patients in whom we have undertaken conservative rather than radical procedures, and after three to five years we have often wished we had done a more radical procedure.

A point of interest we have observed is the apparent better prognosis of cancer of the larynx in women than in men. Lesions along the cord which we would feel require laryngectomy in men, have responded satisfactorily to x-ray therapy or laryngofissure in women. I wonder if Dr. Figi has had a similar experience.

I should like to ask about management of hyperkeratosis of the larynx and whether, in his opinion, the extensive keratotic lesion

should be treated as carcinoma of the larynx or considered a pre-cancerous lesion and kept under observation until it breaks down to a true malignancy.   Another question: Do you do a radical resection of metastatic glands of the neck, or do you treat them routinely by introduction of radon seeds as mentioned in one or two cases?

Dr. Figi stressed suspension laryngoscopy for diagnosis and therapy of laryngeal lesions, and mentioned that this is his method of choice.   It gives an excellent view of the larynx but, as with other surgical procedures, the method of exposures can be left to the choice of the surgeon.   A word regarding x-ray therapy of early lesions of carcinoma of the larynx: Remarkable results have been reported, and we are interested in following our own cases selected for this method of treatment, as well as those of others.   We must watch the results over a period of years and check our statistics not only with regard to three- or five-year survival rates, but for the remainder of the patient's life.

DR. M. F. SNITMAN: I wish to present two questions: What are the results of treatment of advanced lesions of the larynx by laryngectomy and electrosurgery and what, if any, postsurgical irradiation is employed?   During the past few years we have consistently employed a combination of surgery and irradiation and feel that this may be the factor in increasing the cure rates.

No mention was made of transitional epithelial cells and cylindrical cell carcinomata.   Does Dr. Broder place this type in his Grade IV classification?   In the medical literature I have been able to find but one paper which calls attention to the various types of carcinomata arising in different areas of the larynx; that was "Carcinoma of the Larynx" by Max Cutler, published a few years ago in the Journal of the American Medical Association.

Malignancy of the larynx can occur in young people, but is not sufficiently realized.   Recently we observed a young man 25 years of age who had been treated for several months by diathermy for tumor of the larynx; the delayed biopsy disclosed carcinoma. We feel that a better standardization of histology of laryngeal malignancy will give a clearer evaluation of the results of surgery or irradiation.   During the past few years we have been making complete microscopic section of such larynges in order to study the organism and type of lesion.   We hope to present some results of this work in the near future.

DR. ROLAND RUSSELL: I would like to ask Dr. Figi to elaborate on irradiation treatment as given at the Mayo Clinic.   I have

been much disappointed in irradiation in therapy of carcinoma of the larynx.

DR. FREDERICK A. FIGI (closing): I am pleased that Dr. Holinger feels as we do concerning the advisability that the surgeon who performs the laryngeal operation should take the specimen for biopsy himself. It is our conviction that a more conservative operative procedure often will suffice if surgery is immediately carried out by the surgeon who has taken the tissue for microscopic study. I was interested in his comment concerning his tendency to be more radical rather than conservative in dealing with malignant tumors of the larynx. Surgical judgment must, of course, depend upon the experience of the surgeon. Personally, I may lean excessively toward the conservative side, but I am convinced that malignant laryngeal lesions for which laryngectomy commonly is performed often can be adequately removed without sacrifice of the larynx. Extensive highly malignant carcinoma with fixation generally cannot be removed surgically without laryngectomy. However, many malignant lesions of low grade or moderate activity can be eradicated by means of hemilaryngectomy, and the function of the larynx and the integrity of the airway can be maintained. Immediate insertion of a skin graft to line the laryngeal wound will at times greatly hasten the healing process. In other cases, a skin graft is required to correct the cicatricial stenosis resulting from removal of an extensive low grade laryngeal malignant tumor.

The management of metastatic cervical lymph nodes in cases of carcinoma of the larynx depends to a great extent on the activity of the neoplasm and the prognosis in respect to control of the primary lesion. Generally, in cases of high grade malignant tumors of the larynx, irradiation of metastatic lymph nodes will accomplish more than extensive dissection of glands.

Malignant tumors often can be removed from the supraglottic portion of the larynx by suspension laryngoscopy and electrocoagulation more satisfactorily than by laryngofissure. In cases in which such removal is definitely indicated and thoroughly carried out, the prognosis is better than if the tumor were removed through thyrotomy. Review of the results in cases of laryngeal carcinoma in which we have employed an approach through thyrotomy shows the percentage of five-year cures to be 83.6, while the percentage of five-year cures among patients treated by suspension and electrocoagulation has been 91.7.

Although a number of patients whom we have treated with fractional roentgen therapy have become entirely well, the percentage

of cases in this group is low. It must be borne in mind, however, that in all these cases the lesions were of advanced malignancy and the patients were considered to be inoperable. The total dose given in these cases was 7,600 r, administered over a period of 25 days. I do not know the actual technical factors involved.

The question of terminology is well taken. As a matter of fact, one pathologist will designate a lesion as a "Grade IV squamous cell epithelioma;" another may term the same lesion a "transitional epithelioma" or a "lympho-epithelioma." Settlement of this matter must be left to the pathologists. In any event, such lesions generally are not operable unless they are well localized.

Experiences with Histamine in Treatment of Nerve Deafness

BAYARD T. HORTON, M.D.

(Abstract)

The treatment of nerve deafness (which some prefer to call inner ear deafness) is an outgrowth of our treatment of Ménière's disease with histamine, which was begun in 1939. At the time of the first report the statement was made: "Our purpose in presenting this form of therapy is to make available to the medical profession a quick and ready method for controlling the acute symptoms in Ménière's disease." While we were concerned primarily with the relief of vertigo, it was noted that the tinnitus and hearing of some patients improved with histamine therapy but the full significance of the latter observation was not fully appreciated. Again, in a report in 1941 I casually mentioned the fact that of 49 patients with Ménière's disease, a small number reported improvement in hearing. Again in 1944, Lillie, Thornell and I reported observations on the hearing of patients treated with histamine and noted that of 25 cases reported "improvement in the deafness was noted in 12 cases."

Yet it was not until March, 1945, that treatment of nerve deafness was attempted. A man aged 45 years came to us because of sudden and complete deafness in the right ear of 24 hours' duration. He did not have vertigo, nausea or vomiting. Histamine (a 1:250,-000 dilution) was administered intravenously, daily for 12 consecutive days, according to our standard method. At the end of that time audiograms showed normal hearing in the right ear and it has remained normal. We have not observed spontaneous recovery of hearing in a case of this type. Stimulated by this case, we have treated approximately 293 patients who have had nerve deafness.

In the past there has been no satisfactory treatment for nerve deafness. As we have previously stated, we believe the primary pathologic lesion to be edema, not hemorrhage, of the cochlear portion of the labyrinth. The edema causes deafness by pressure on the organ of Corti or some interference with the fluid metabolism of the labyrinth. We are obviously dealing with a phase of Ménière's disease. The factor most likely responsible for Ménière's disease is local alterations in the permeability of the capillary wall with secondary local edema. Vertigo is the essential feature and represents a local process involving the labyrinth. The presence of tinnitus and varying degrees of deafness indicates that the process also involves the cochlea. It is likely that in many patients progressive deafness without obvious cause is the result of primary cochlear involvement and that the condition corresponds to that of patients who have Ménière's syndrome, who have recurrent severe vertigo but in whom tinnitus or loss of hearing never develops.

In this report no attempt will be made to survey the literature but certain significant observations made by others will be referred to briefly. Dederding in 1929 advanced the theory of disturbed water metabolism as the etiologic factor in Ménière's symptom complex. Later Mygind and Dederding postulated that the deafness which resulted was an inner ear conduction type of deafness. By reducing the fluid intake together with other general supportive measures they reported that in 85 of 150 cases the hearing improved; in 47 cases the hearing returned almost to normal and in 24 cases there was no improvement.

Lindsay in 1942 reported the micropathologic observations in a case of bilateral deafness in which there was evidence of greatly increased endolymphatic pressure. He said that such a case can be classified under Ménière's symptom complex. To him, increased endolymphatic pressure provides the most rational basis for the explanation of this symptom complex. The auditory symptoms he explained on a physical or mechanical basis; that is, an interference in the transmission of sound in the cochlear fluid is the result of a distorted saccule, Reissner's membrane, and disproportion of both the scala media and scala vestibuli.

The term "nerve deafness" as used in this discussion is not meant to imply that irreversible degenerative changes have occurred in the cochlea or in any portion of the eighth nerve. It is meant to indicate the same fundamental disturbance as that discussed by Mygind, Dederding and Lindsay as inner ear deafness. More adequate terminology may eventually evolve from the work in this field when the exact nature of the disturbance is determined.

If degenerative changes have occurred, treatment will be of no avail. However, I am of the opinion that the fundamental pathologic process which accounts for deafness in Ménière's symptom complex accounts for loss of vision in idiopathic retrobulbar neuritis. Defects in the hearing range in subjects with Ménière's symptom complex have their counterpart in similar defects in the perimetric fields of subjects with retrobulbar neuritis. Edema occurs in both instances in areas in which there is little or no room for expansion; this results in the classic signs and symptoms which characterize Ménière's symptom complex and retrobulbar neuritis. It is well known that a patient with retrobulbar neuritis may have complete loss of vision in one or both eyes and still make a complete recovery. Ophthalmologists are not in the habit of thinking in terms of degenerative changes at the onset of the disease. Optic atrophy may occur later and frequently does. The same line of thought applies equally well to the auditory nerve and to the cochlea. The sudden onset of tinnitus in one or both ears which has been noted to occur simultaneously with the development of new hemorrhagic areas in a patient with bilateral chorioretinitis, makes one think that the same disturbance seen in the fundi may also be occurring in the labyrinth. The simultaneous disappearance of tinnitus and retinal hemorrhages makes this assumption seem probable.

Since we were finally stimulated early in 1945 to begin treating the deafness which is so frequently observed in subjects with Ménière's symptom complex, we have had occasion to treat 293 patients. Treatment in most instances was directed primarily toward improvement of hearing. In many instances vertigo was present, but even though this was relieved we continued with the treatment.

Since we began giving histamine intravenously in 1939, approximately 75,000 such injections have been given. It continues to be a simple procedure which can be carried out in the office. To prepare the solution, 2.75 mg histamine diphosphate (1 mg histamine base) is diluted in 250 cc of physiologic saline solution. This is administered by the drip method at rates usually ranging from 16 to 60 drops per minute, depending upon the tolerance of the individual. It is the rate of administration rather than the amount of solution which is of paramount importance. At no time is the treatment continued for more than one and one-half hours; no more than 250 cc of solution is administered at any one time. The procedure is carried out daily for an indefinite period, dependent upon the individual response. The rate of administration is increased gradually, but is always kept below the reaction level; that is, the individual should not experience headache or any other discomfort

during treatment. The patient should have food in his stomach at the time of administration so that excess gastric acids provoked by the solution will be absorbed promptly. Reactions do not occur when the procedure is carried out properly. Such reactions are not due to histamine itself but rather to the presence of pyogenic substances in the tubing or equipment used. Disposable tubing should be employed to avoid such reactions.

The literature concerning histamine is confusing to the average reader. It has often been assumed that the physiologic effects observed in experimental animals also occur in human beings. In man, histamine dilates the arterioles, venules and capillaries. The rationale for its clinical use is chiefly dependent upon this fact. It is not only a powerful dilator of capillaries but also increases capillary permeability. Its effect upon the arterioles varies with the animal species concerned. For example, the arterioles of the cat are constricted slightly while those of the dog, monkey and man are dilated by it. The possibility that histamine ever constricts smooth muscle in normal human beings is extremely doubtful.

It is on the basis of the many physiologic effects of histamine that it has been used clinically in treatment of deafness. Of the 293 patients treated, 75 were treated during 1948 and these results have not been tabulated; of the remaining 218, 54 were eliminated because of inadequate data. In 164 cases which could be evaluated, 16% obtained good to excellent restoration of hearing; 41% noted some improvement; 32% did not obtain any improvement in hearing; 11% did not have adequate treatment. Two factors seemed significant in the results: (1) duration of hearing loss; (2) duration of treatment. As a rule, two to three weeks or more of treatment are necessary before a change is noted in the hearing. Treatment is continued as long as the audiograms show improvement. In some instances this may mean that treatment is continued for several months.

DISCUSSION

Dr. Francis Lederer: This is rather paradoxical; I should have discussed the previous paper on laryngeal carcinoma. I know nothing about this except from clinical experience. I am afraid we are all going to sing, "Roll out the barrel and bring in the histamine." There is a danger in speaking of a treatment of nerve deafness in general terms, and I think Dr. Horton and his associates will have to qualify the type of case suitable for this type of therapy, and give us clinical tests and therapeutic tests under which this agent is employed. When one considers the number of case reports on

prostigmine and thiamin chloride treatment—all most enthusiastic— one is inclined to feel that unless this treatment can be carried out as it is by Dr. Horton, it should not be done.   I have not seen a clear cut picture demonstrated tonight, and I am rather concerned that the essayist, it his excellent presentation, has left us without all of the facts, and that we may be too prone to accept this agent.   I have seen similar results, such as he has shown on the screen, from other methods.   As a matter of fact we have observed improvement from antihistaminic agents.   In this same Society we have heard of a successful treatment of vertigo and an improvement of deafness and tinnitus by the use of pyridoxine.   All I want to say is that I thoroughly appreciate the paper and the presentation but, at the same time, let us not treat every person with so-called nerve deafness, until we know what cases should be treated by this agent.   Indiscriminate application of histamine should be discouraged and a better selection of cases arrived at.

DR. ALFRED LEWY: I notice that Dr. Horton's patients, with two or three exceptions, were mostly young people.   This may have a great bearing on treatment of nerve deafness.   Most patients I see with nerve deafness are beyond middle age, and I must confess that I do not know how to cure nerve deafness in middle age.   On the other hand, young people respond to treatment based upon whatever deficiency has been discovered.   However, I have not seen any such outstanding improvement as Dr. Horton shows on the screen, but I have seen considerable improvement in young people.

Dr. Lederer mentioned the use of pyridoxine with reference to vertigo, particularly associated with Ménière's syndrome.   Pyridoxine has the advantage of much greater simplicity of administration and greater range so far as results are concerned; it requires no hospitalization.   I did not note any consistent improvement in hearing in cases of Ménière's syndrome.   This may be due to the fact that patients who had improvement from vertigo did not have a long enough time for improvement in hearing to be observed.   If I can persuade my patients to try pyridoxine over a long period of time, perhaps better results in hearing may be obtained.

DR. JOHN R. LINDSAY: The remarks I have to make and the questions I have to ask probably should be directed to Dr. Horton's associates in this presentation since they concern diagnoses in the cases shown.   Several of these cases were labelled Ménière's syndrome, Ménière's disease or Ménière's disease without vertigo, or hydrops without vertigo.   The available evidence indicates that in order to make the last named diagnosis, you must have a characteristic type

of deafness, and the patients must be observed for a sufficient period to determine whether they have the characteristic threshold variations. From the evidence presented on the screen, the diagnosis of hydrops is open to question in some cases. For example, an impairment of hearing which had existed for only a few days without vertigo could not reasonably be considered as hydrops without vertigo. Also one must question one of the audiograms which I believe showed loss of air and bone conduction beyond the 100-db level. Also a total loss of unilateral hearing due to uncomplicated idiopathic hydrops is a diagnosis not easily supported.

I have enjoyed Dr. Horton's presentation and would like to think that he has presented something that will offer some hope for a better method of treatment than we have had until now.

DR. J. A. WEISS: In order to obtain a more accurate definition of the type of cases in this report, I would like to ask Dr. Horton if he has any data as to the incidence of clinical allergy in his series.

DR. BAYARD T. HORTON (closing): I appreciate the opportunity of appearing before this Society as a guest speaker. The observations I have presented regarding treatment of nerve or inner ear deafness represent investigative work done in conjunction with members of the Department of Otolaryngology at the Mayo Clinic. The diagnoses and the audiograms were made in that department and treatment was carried out in my laboratory. Patients with psychogenic deafness were excluded from the study.

I am aware that the improvement in hearing demonstrated seems spectacular. However, I have presented facts as we have observed them. I am not concerned about the skepticism that has been manifested. Histamine therapy for nerve or inner ear deafness does not have to be defended. If there is any virtue in it, it will live; if not, it will die.

Meeting of Monday, February 7, 1949

THE PRESIDENT, DR. WILLIAM A. SMILEY, IN THE CHAIR

DR. FRANCIS LEDERER: The work to be presented tonight by the group from the Veterans Administration at Hines covers 1325 operative procedures performed by members of the Staff, and represents many interesting phases of otolaryngology. Otolaryngology is being recognized as a head and neck specialty and is well supported at the Veterans Hospital. We feel that you will be interested in this

report to the Society, as the group is really part of the Society and we have many veterans here, and many members of the Deans' Committee at Hines.

Rehabilitation Surgery in Gunshot Wound of the Neck

STANTON A. FRIEDBERG, M.D.

This report deals with the rehabilitation of a wounded soldier. Although there are decreasing numbers of men with service-connected surgical disabilities still under treatment at Veterans Facilities, this particular case history doubtless has its counterpart in many other hospitals throughout the country. It is presented in order to illustrate one aspect of veterans' care which inevitably receives less notice with the passage of years following a war.

The patient, a 37-year-old infantryman, was wounded during the fighting in the Siegfried Line on February 3, 1945, receiving injuries in the neck and larynx from a shrapnel fragment. An immediate tracheotomy was performed at the battalion aid station and he was evacuated through various channels until he reached Hines Hospital on May 15, 1945.

The first Hines Hospital admission indicated the portal of wound entry to have been the left hyoid region; the point of exit was in a comparable location on the opposite side. Both sites were healed and the tracheotomy was functioning well. There was paralysis of the left hypoglossal nerve and marked scarring of the posterior pharyngeal wall. Distortion of the larynx obscured detailed examination so that the vocal cords were not seen. Roentgenograms of the neck showed some fragmentation of the hyoid bone and of the thyroid cartilage. There were no foreign particles. The patient was discharged on June 3, 1945, without further treatment.

He was re-admitted on February 4, 1946, at his own request, for removal of the tracheotomy tube. A report of a direct laryngoscopy at this time mentioned strictures of the aryepiglottic folds and pyriform sinuses, with fixation of the right vocal cord. The tracheotomy tube was removed but had to be replaced after several days. Closure of the tracheal stoma was thus deemed inadvisable and the patient was again released, this time with recommendation for follow-up examination in six months.

He again entered the hospital in February, 1947, just two years after the original battle injury. Two months previously the wound

in the left neck had begun spontaneously to discharge clear fluid. Shortly thereafter ingested liquids escaped through the opening. Difficulty in swallowing any sizeable food bolus was mentioned. The voice was quite rough. Mirror laryngoscopy disclosed marked distortion of the pharynx extending inferiorly from the base of the tongue. Extensive irregular scar tissue and a series of step-like webs produced a funnel-shaped lumen which narrowed down to a diameter of approximately 1.5 cm in the hypopharynx. Because of this, no satisfactory evaluation of the larynx could be made with the mirror. The strictured pharyngeal passage was dilated with a small anterior commissure laryngoscope until the larynx came into view. It then became evident that a scar tissue web occupied the greater part of the glottic space, with only a small horizontal aperture remaining posteriorly. There was complete fixation of the left vocal cord and limited motion of the right. As the laryngoscope was removed, a probe introduced into the cervical fistula could be seen to emerge in the central portion of the scarred hypopharynx. Roentgenograms of the chest, serologic tests and routine laboratory studies were negative.

It was felt that an attempt should be made to repair the pharyngeal fistula before undertaking restoration of laryngeal function. The extensive mucosal scarring in the pharynx was believed to present an obstacle for satisfactory closure and plastic surgery consultation was requested.

The operation upon the pharyngeal fistula was performed on May 14, 1947, by Dr. C. W. Monroe, with the essayist assisting. General anesthesia was employed. A small probe was sutured into the fistulous tract and the latter was dissected out along with considerable surrounding scar tissue. A portion of the submaxillary gland was involved in the tract and was removed during the dissection. The pharyngeal defect was closed with interrupted dermalon sutures and reinforced with No. 00000 chromic catgut. A feeding tube was introduced, the remainder of the submaxillary gland and surrounding scar tissue were removed and small rubber drainage tubes were inserted during closure. Because of extensive fibrosis this procedure involved five hours of painstaking dissection. Primary healing resulted.

Eight weeks later the laryngeal web was excised and laryngeal dilators through size 24 were easily passed. Dilatation was carried out at weekly intervals thereafter. This procedure had an added beneficial effect in that the repeated laryngoscopies served to increase the diameter of the narrowed hypopharynx. Six weeks later there was free mobility of the right vocal cord, some limitation of

movement in the left cord, and a sizeable glottic aperture with epithelialized cord margins.   The tracheotomy tube was completely corked for two weeks.

On August 22, 1947, under local anesthesia, closure of the tracheostomy was undertaken.   A quadrangular series of connecting incisions was made 4-5 mm above and below the stoma.   After undermining the skin almost to the edges of the fistula, the lateral margins of the prepared flaps were inverted and sutured in the midline with fine catgut.   This resulted in an epidermal lining for the tracheal lumen and a subcutaneous layer of tissue externally.   Two sliding skin flaps of unequal length were developed and brought together so that the line of closure would not fall directly over that of the fistula.   Care was taken to avoid tension in the wound closure and several lengths of silkworm suture were laid horizontally along the entire length of the field for drainage.   The wound healed primarily.   There was no evidence of a narrowed airway.   The voice was rough but of adequate intensity.   The patient left the hospital two weeks after operation or almost five months after admission. He has returned to his prewar job in a paint shop and, despite rather strenuous work, has had no difficulty in performing his duties. His only complaint is inability to raise his voice above the din of particularly noisy surroundings.

The breakdown of pharyngeal scar tissue with resulting fistula two years after the original injury merits comment.   It is felt that the increased propulsive efforts required for swallowing in a fibrotic, narrowed hypopharynx may have been responsible for this. Partial removal of scar tissue and dilatation augmented the caliber of the pharyngeal lumen.   In all probability the glottic area was not involved in the penetrating wound, the greater portion of the thyroid cartilage having remained intact.   More extensive surgery for establishment of an airway would have been necessary had there been greater damage to the body of the thyroid cartilage.   Release of intracordal scar tissue resulted in free mobility of one cord with partial motion of the other, and dilatation was effective in maintaining patency until healing occurred.

The method employed for closure of the tracheostomy is applicable only to relatively small tracheal defects.   Larger openings may require the use of skin flaps in which have been incorporated bone or cartilage implants for maintenance of the tracheal lumen.

DISCUSSION

DR. JOHN J. BALLENGER: When I was overseas I saw a case somewhat similar to this.   Of course we could not keep track of

cases at that time. We thought we could prevent this cicatricial contraction of the larynx by an acrylic implant, and we inserted this and sent him on his way about three weeks later.

DR. FRANCIS LEDERER: The tracheotomy was always our difficulty in war casualties. The placement of a tracheostomy was never uniform. One always feels that hindsight is better than foresight, and when we finally got these cases after they had gone through various centers, we could not be too critical. However, it did seem that better tracheotomies should be taught the medical officers, as well as those in civilian practice.

DR. STANTON A. FRIEDBERG (closing): I should like to thank Drs. Ballenger and Lederer for their discussion. As a rule the acrylic implants mentioned by Dr. Ballenger are used in the later stages of reconstructive surgery of the larynx. It would be interesting to know how these implants affect the progress of events when used immediately in laryngeal trauma. It is entirely possible that they might provide a lumen which would heal without the formation of scar tissue contractures.

Management of Advanced Cancer of the Larynx

MAURICE F. SNITMAN, M.D.

Malignant disease in the larynx can be divided into two groups: A., the generally curable; and B., the generally incurable. The former can be subdivided into two groups; the early cordal lesion amenable to laryngofissure, irradiation therapy and not infrequently eradicated by excision biopsy. Pathologists have expressed the opinion that a simple stripping of the mucosa would cure such cases. The response of this group to almost any type of therapy is so good that it merits no further consideration. Group 2 includes more extensive endolaryngeal lesions that can usually be cured by either laryngofissure or laryngectomy, and in some cases by irradiation.

Group B includes the so-called advanced and inoperable carcinomas of the larynx. This group may also be divided into three subgroups:

B_1: With no cervical metastases but with primary lesion spread to the pharynx;

B_2: With operable cervical metastases;

B_3: With residual after complete course of irradiation.

Statistic conscious therapists vie for Group A_1 and for the more select cases in A_2. Concentration upon this group does little more than inflate the ego of the therapist. The problem to be faced is that of Group B. This group includes at least 75% of the patients diagnosed as having carcinoma of the larynx. The prolongation of life and alleviation of distressing symptoms in this group is the responsibility of the cancer therapist.

In assessing the value of treatment of such cases it is essential to distinguish between immediate effects and ultimate results. Ultimate results as reported have been universally poor (5-10% cures) whether treated by irradiation or surgery. Reports of the combination of both modalities are very scant and do not supply the clear-cut description of the technique and the factors of such therapy, to say nothing of their results. When applied in terms of a five-year cure rate, we are not as yet in a position to offer statistics over such a period. We are more concerned with immediate effects and the hope of increased salvage of such cases so as to increase the ultimate results.

During the past 18 months we have modified our standard of operability.

Group B_1:   Those with no clinical evidence of lymph node metastases, but where the primary lesion of the larynx has spread to the pharynx, or broken through the laryngeal cage to involve the extralaryngeal structures. If grossly the lesion can be circumscribed by a total laryngectomy, or combined if necessary with a pharyngectomy, such a case is considered operable. Postoperatively a cancerocidal dose of external irradiation is applied.

Group B_2:   Same as Group 1 but with operable cervical nodes. The larynx removal with or without a pharyngectomy is dictated by extent of the lesion, followed in ten days to two weeks by radical neck dissection, to be followed by a full course of x-radiation several weeks later.

Group B_3:   It has been our experience on repeated occasions to observe patients who evidence local residual carcinoma months or years after a complete course of irradiation. Total laryngectomy performed in such cases is attended by no appreciable alteration of the surgical procedure or the eventual postoperative progress.

The broadening of the field for surgical intervention and a closer co-operative effort with the radiotherapist has several advantages. The salvaging of some cases, even a few, more than justifies the effort. Prolongation of life is difficult to estimate and impossible to prove; nonetheless we are of the opinion that we have lengthened

some lives. The alleviation of distressing symptoms has contributed to the comfort of those who could not be cured.

In conclusion, we recommend to the laryngologist a greater concentration and effort in the less hopeful cases of carcinoma of the larynx, to the end that we may achieve more life, more years, and more comfort for our patients.

DISCUSSION

Dr. JOSEPH G. SCHOOLMAN: I would like to compliment Dr. Snitman and the group at Hines for their work in this field. Those of us who follow cancer patients—and we tell them we will follow them the rest of their lives—realize the disheartening period so many of them may go through. Procedures of this kind, which can show results in only a relatively small group of cases, do not mean a whole lot from the statistical point of view, but from the point of view of the patients it is most important.

Dr. FRANCIS L. LEDERER: We have reached the period in life with respect to cancer where such a formidable enemy as confronts us in cancer of the larynx requires all-out effort on our part. The so-called Group A cases are generally amenable to irradiation or to operation. The importance of Group B cases is to be emphasized. This group should occupy a good part of our attention. I, for one, am willing to disregard statistics. If I have contributed to the rapid passing of some of the patients I have had to do with under these circumstances, I am perfectly willing to take the responsibility, because I do not think this problem will be solved merely by admitting that such cases are inoperable.

Dr. Figi, at the last meeting of this Society, said one could only conclude that all factors covering surgical and medical treatment must be known to the laryngologist. The radiologist is not particularly enthusiastic about treating any case we deem inoperable, but surely a combined attack, using both modalities, may be responsible for salvaging a few of these cases, and may be well worth while.

Otologic Effects of Streptomycin

(Supplementary Report)

LINDEN WALLNER, M.D.

In February, 1948, I presented before this Society a report of ear examinations performed on 93 patients who received strepto-

mycin for 120 days. Of those who received 2 gm per 24 hours, 47% had objective evidence of vestibular damage; 32.5% of those who received 1 gm per 24 hours had signs of impaired function. At that time it was stated that the dose was being reduced to 0.5 gm per day in the hope of reducing the toxic effects.

This is a report on 66 patients who have received the 0.5-gm dose for the treatment of tuberculosis, at Hines Hospital. Thirty-nine patients received the 0.5-gm dosage for 120 days. Later the drug was given for 42 days, and 27 received it for this shorter period.

Tests on the ear were carried out as in the previous report. The hearing was tested on the audiometer before, during and after treatment. Caloric tests were also done before, during and after treatment by the Kobrak method as modified by McNally. Subjective complaints relative to the ear were noted. Some of the previously reported patients with vestibular damage were still available for further study. Tests were done to determine if there was any late return of function in those who had shown depressed or absent caloric responses.

RESULTS

1. Subjective complaints: Only 7 patients admitted on questioning that they had noticed some vertigo while receiving the drug. It was severe enough to affect walking in only two patients. This lack of vertigo, tinnitus and blurring of vision was in sharp contrast to the large number of patients who had complained of such symptoms while receiving the larger doses.

2. Hearing: None of the patients had any loss of hearing, as revealed by audiograms made before and after treatment.

3. Caloric tests: Only two of the 66 patients (3%) had significant changes in the caloric responses after treatment. These were the two patients who had complained of severe vertigo. Each had absent caloric response in one ear, noted one month after starting the drug. This absent response in one ear has persisted to the present time, though neither notices vertigo now, or has any trouble in walking.

4. Follow-up of previously reported patients: No patient with absent caloric responses revealed a return of function when tested later. With some, two years have elapsed since receiving the drug.

COMMENT

While 2 of 66 patients, or 3%, had objective evidence of damage from the 0.5-gm dose, in each only one ear was involved. They

do not have difficulty in walking comparable to those with bilateral damage. The 0.5-gm dosage is thus seen to be relatively much less toxic to the ear than 1 or 2 gm a day. The first question then would be whether this smaller dose is as effective therapeutically. In observing the effects of the drug in laryngeal tuberculosis it was my impression that the larger doses acted more dramatically. Levine, Klein and Froman state that the 0.5-gm dose is adequate, yielding as good results in pulmonary tuberculosis as larger doses. This is also the opinion of most of the men on the tuberculosis service with whom I have talked. Two or three grams per 24 hours is often given for seven to ten days for tularemia, kidney infections and other diseases due to gram-negative organisms. If the safer 0.5-gm dose is effective in tuberculosis, it might also be as useful in other infections. The newer drugs, dihydrostreptomycin, aureomycin and chloreomycetin are said to be less neurotoxic and just as effective as streptomycin, and may provide the solution of the problem.

It is often said that patients soon recover from or compensate for vestibular damage due to the toxic effects of streptomycin. It is true most of the patients do not complain of dizziness one or two years later, and state that they have less trouble walking. Two patients with absent caloric responses whose gait was recorded in moving pictures one year ago were again photographed. They are still seen to walk with the feet wide apart, obviously relying on visual aid. When blindfolded they are quite as ataxic as they were a year ago.

CONCLUSIONS

The toxic effects of streptomycin on the inner ear are serious in nature. When once present, they seem likely to be permanent. The 0.5-gm dose seems a nice compromise; it is relatively nontoxic to the inner ear, yet is effective therapeutically.

DISCUSSION

DR. SHERMAN L. SHAPIRO: I do not know whether I was one of those who last year thought the toxic effect would be temporary. I certainly do not think so now. This presentation, which we all hope will be just a passing landmark in medicine, that the newer drugs will avoid these distressing symptoms, does point to a basic fact in medicine. We all know from observation of tabetics in days gone by, that at least two of three essential senses, vestibular sense, vision and tactile sense, are necessary if station and locomotion are to

be effective. Even a fish, if deprived of its labyrinth, will swim upside down until it reaches the side of the tank, whereupon its tactile sense comes into play, it turns over and swims upright.

I wonder whether these experiments had been carried out on cases of congenital or acquired deafness. It seems to me that the school of Jacksonville might be a good place to try this, to see whether some of the children react in the same way. I think they would. I think we are dealing with the basic fact that an individual cannot operate successfully, especially with the tactile sense alone.

DR. ELMER W. HAGENS: Some years ago we examined children at Jacksonville, as well as other children in state schools for the deaf. As I saw the movie, I wondered why we had not noted staggering in these children. We did not blindfold them, but certainly they had no such complaints as these patients who had streptomycin. So far as I know, it is believed that the pathology in streptomycin cases is involvement of the vestibular nuclei at the base of the brain. In the children at Jacksonville the pathology is peripheral. In meningitis it is a nerve labyrinth affair. It may thus be that the pathology in the streptomycin cases involves the basal nuclei, and in the deaf children it is peripheral. It occurs to me that we might investigate the children in the schools to see if there is any comparison or anything of contrast. One would think, if these patients had no response to caloric tests, that they would get over this. We have always thought there was compensation, not that they could walk well in the dark, but that they should not stumble as these streptomycin patients do.

DR. MARVIN TAMARI: I think this film shows very nicely ataxia as seen in cerebellar lesions. It is known that after surgery of the labyrinth or fenestration patients do not stumble nor show ataxia after such a long period. Those patients shown in the film, in my opinion, show central lesions but not peripheral. In the postmeningeal cases at the Jacksonville school we have not seen this type of ataxia, although there are some balance difficulties, especially in extending the head backward and in darkness.

DR. FRANCIS L. LEDERER: You will recall that some of these cases were influenced by pyribenzamine. Walking with the broad base gave evidence that this was not a peripheral disturbance. In postmeningitic cases there may be certain difficulties for a time, with later compensation, but these patients in the streptomycin group have not recovered their equilibrium.

DR. LINDEN WALLNER (closing): I cannot answer the question why deaf children with absent caloric responses do not have

similar difficulty in walking. It could be the earlier age at which they lost vestibular function. Also the lesion is peripheral, usually, while in streptomycin toxicity it is thought to be central.

Some of these patients have been seen by neurologists, but I do not have their reports available.

Abstracts of Current Articles

EAR

Treatment of Ostitis Temporalis Chronica by Simple Chiselling, Sulfathiazol Plombage and Primary Suture.

Norup, Svend: Acta Oto-laryngologica 36:7-15, 1948.

Sixty cases of chronic otitis media with mastoiditis were treated by simple mastoidectomy, 28 cases showed marginal perforations, 32 cases, central perforations. Shorter convalescence, fewer continually discharging ears and better cosmetic results are reported as a result of this procedure. While the advantages of primary suture over packing are admitted, there might be considerable doubt as to the beneficial part played by the local use of sulfathiazol. One might likewise hesitate to rely on simple mastoidectomy in all cases showing marginal perforations.

PRATT.

Attic Cholesteatoma Following Longitudinal Fracture of the Pyramid.

Nilsson, Gunnar: Acta Oto-laryngologica 36:85-91, 1948.

A case of attic cholesteatoma is reported following longitudinal fracture of the petrous pyramid. The mastoid cells were normal. It is hypothesized that this arose from the healing of the ruptured tympanic membrane with invasion of the middle ear with epithelium.

PRATT.

Cerebrospinal Meningitis and the Acoustic Function.

Johnsen, S.: Acta Oto-laryngologica 36:169, 1948.

In cerebrospinal meningitis the labyrinth may become involved via the perineural spaces around the acoustic nerve or along the cochlear aqueduct, resulting in perception deafness. Prior to the use of chemotherapy this occurred in about 7% of cases. In 23 patients who had chemotherapy, only one showed hearing impairment which could be attributed to the meningitis, while 4 out of 10 patients not so treated, had marked impairment.

HILL.

Labyrinthine Surgery for Ménière's Disease.

Lindsay, J. R.: Laryngoscope 59:22-34 (Jan.) 1949.

Lindsay describes and evaluates various surgical measures advocated for treatment of Ménière's disease. These procedures are adopted as a last resort and directed primarily at the prevention of vertigo. Improvement in hearing is not expected and in some cases deafness becomes complete and permanent following surgical procedures.

The author determined by experiments on monkeys that drainage of the saccus endolymphaticus such as advocated by Portmann would have only temporary effect. Alcohol injections are not advocated; although favorable as regards vertigo, tinnitus remains and deafness always follows. Removal of the ampullary end of the membranous horizontal canal along with its ampulla (Cawthorne) prevents vertigo but results in deafness in the operated ear.

Day reports preservation of useful hearing in 10% of cases, with relief of tinnitus and vertigo, by coagulation of the labyrinth. He coagulates the horizontal membranous canal, ampulla and the vestibule through a small fistula near the ampulla. Other surgeons have obtained less favorable results.

This procedure according to Lindsay offers the most encouragement of the advocated procedures for Ménière's disease. Further investigations with improvement in technique may bring us nearer to the goal of prevention of vertigo and tinnitus with preservation of hearing.

VAN ALYEA.

Practical Aspects of Labyrinthine Tests.

Hoople, Gordon D.: Laryngoscope 59:12-21 (Jan.) 1949.

Hoople describes in detail the Hallpike-Cawthorne method of labyrinthine testing. This method, he maintains, supplies all information obtainable by the other procedures; yet is devoid of undesirable side effects.

In this procedure the patient is prone with the head elevated at an angle of 30 degrees. Each ear is tested with both hot and cold water at 44° and 30° C. respectively. The testing fluid is placed in an irrigating can some two or three feet above the head of the patient, and, with as little waiting period as possible, fed to the ear through a rubber hose and appropriate nozzle. Prior to each douching, the water in the tubing is run off and discarded in order that the proper temperature be maintained. The douching period in

each instance is 40 seconds and time is measured from the beginning of the douching up to the end of nystagmus. This duration time is noted and charted in what the authors call a calorigram. During the period required for observation of the duration of the nystagmus the patient is instructed to look at a convenient mark on the ceiling directly overhead.

The procedure is carried out without the use of Bartel's glasses or Frenzel's lenses for it is felt that they prolong the reaction or diffuse the end-point. While attention is principally devoted to the duration time, the authors note that the degree of intensity of the nystagmus and the patient's subjective reactions (dizziness) should be recorded. A small difference in duration time between the two ears is of much more significance if it is associated with an obvious difference in intensity of nystagmus and dizziness.

The method is simple and not unpleasant to the patient and a test which more nearly approaches standardization than the others.

VAN ALYEA.

Sudden Deafness.

Rasmussen, H.:Acta Oto-laryngologica 37:65 (Feb.) 1949.

Eighteen cases of sudden deafness, in 15 of which there were vestibular disturbances, are reported. Three were considered as due to thrombosis or hemorrhage in the labyrinth, one to vascular spasm and the remainder to neuritis of the auditory nerve.

HILL.

Histological Investigation of Three Cases with "Ménière" Syndrome.

Berggren, S.: Acta Oto-laryngologica 37:30 (Feb.) 1949.

The author mentions a case previously reported and two additional ones in which microscopic examination of the labyrinth failed to reveal abnormalities. The only abnormal finding in the brain was numerous dilated blood vessels in the floor of the fourth ventricle. He feels that the condition may be due to a central vascular disorder. However, it seems unlikely that the author was dealing with true Ménière's disease as the signs and symptoms reported would not be sufficient to warrant a diagnosis of labyrinthine hydrops.

HILL.

Otitis Media Following Electrocoagulation of the Gasserian Ganglion after the Method of Kirschner.

Johnson, S.: Acta Oto-laryngologica 37:45 (Feb.) 1949.

In a study of 74 patients with trigeminal neuralgia, treated with electrocoagulation of the ganglion, 10% developed suppurative otitis media in the homolateral ear and 20% complained of symptoms of tubal obstruction. The author attributes this to paralysis of the motor root with consequent involvement of the tensor veli palatini muscle and resulting tubal occlusion.

HILL.

Sur la chirurgie de l'otospongiose.

Sourdille, M.: Acta Oto-laryngologica 37:195 (June) 1949.

The author states that 50% of fenestra will shrink or close despite whatever method be employed to create the opening into the labyrinth and that re-operation should give better than 80% of successful results. He advocates conservation of the incus, complete mastoidectomy and the postaural approach using a two-stage operation. He makes a plea for the divorcing of otology from rhinolaryngology in order that otologists may devote their efforts better to the problem of surgery of deafness.

HILL.

Penicillin Treatment of Simple Acute Otitis Media.

Riskan, N.: Acta Oto-laryngologica 37:230 (June) 1949.

One hundred and thirty-three patients treated with penicillin were compared to 127 treated with irrigation. While mastoidectomy was not required in the penicillin-treated group and was necessary in 20 of the controls, and the suppurative stage of the former averaged 7.1 days in contrast to 13.2 in the latter, it was felt that penicillin therapy increased the chance of recurrence and of new infection. Cases due to Pfeiffer's bacillus did not respond. It was felt that penicillin therapy should be restricted to hospital and complicated cases due to other than Pfeiffer's bacillus.

HILL.

Primary Reduction of a Large Operative Cavity in Radical Mastoidectomy with a Muscle-periosteal Flap.

Meurman, Y., and Ojila, L.: Acta Oto-laryngologica 37:245 (June) 1949.

The author presents a technique for obliterating defects in the tip in cases of extensive pneumatization. A flap is formed from the attachment of the sternomastoid muscle and its periosteal extension, and placed in the lower portion of the operative cavity. The method is applicable to either the postaural or endaural operation.

HILL.

The Passage of Fluoriscin Sodium to the Labyrinthine Fluids.

Gisselsson, L.: Acta Oto-laryngologica 37:268 (June) 1949.

In an experimental study using guinea pigs and cats it was found that fluoriscin injected intravenously passed rapidly into the cerebrospinal fluid but did not appear in the perilymph for several hours unless the labyrinth was punctured to allow free escape of perilymph. It was felt that this work corroborated the theory that perilymph is derived from the cerebrospinal fluid while endolymph is formed by secretion in the membranous labyrinth. In pathological conditions the endolymph may diffuse or filtrate into the perilymphatic space.

HILL.

NOSE

Penicillin Treatment of Acute or Subacute Maxillary Sinusitis.

Gording, R.: Acta Oto-laryngologica 37:12 (Feb.) 1949.

Sixty cases of acute and subacute maxillary sinusitis were carefully studied as to the bacteriology. Approximately half of these showed mixed infection. The duration of treatment varied greatly regardless of the nature of the infection. Thirty-two patients did not have penicillin therapy while 28 were treated locally and parenterally with penicillin. While the number of cases was too small to draw conclusions it was felt that penicillin therapy as employed failed to shorten the period of treatment. Heavier dosage in the early stages might be more effective.

HILL.

Constitutional Effects from the Use of Sympathomimetic Drugs As Nasal Medication in Children. Report of Case of Privine Toxicity.

House, Leland R., and Carey, Wells, C.: Laryngoscope 58:1294-1298 (Dec.) 1948.

Privine hydrochloride when applied locally is capable of producing a prompt and thorough nasal vasoconstriction. But when used for any length of time, the patient becomes dependent upon the drug and this dependency may last indefinitely.

Aside from this local reaction, House and Carey have noted systemic toxic effects, especially in children, when a few drops of privine are instilled in the nose or swallowed. They present one case and cite four from the literature in which there is a period of stimulation followed by depression due to the medication. All cases recovered a few hours after elimination of the drug.

VAN ALYEA.

Clinical Study of Vonecidin.

Calvin, Davis L.: Laryngoscope 59:272-277 (Mar.) 1949.

The ideal vasoconstrictor for the nasal mucosa should provide effective and prolonged vasoconstriction without disturbing the normal physiology of the nose. It should be nonirritating and should not cause a secondary congestion or turgescence of the nasal mucosa.

Vonedrine exerts its vasoconstrictor action on the deep vascular plexus. It causes effective decongestion without blanching the superficial mucosal vessels, without stinging or burning. The onset of the vasoconstriction is not as rapid as that of some other vasoconstrictors but its action is more lasting. It is not followed by any secondary congestion. It has a low toxicity.

Davis has used vonedrine in a 2.8% solution in packs and sprays and finds that it produces desired results without any toxicity and without producing nervousness or a rise in blood pressure and, therefore, it has a distinct advantage over ephedrine solutions.

Davis calls attention to vonedrine solutions fortified by an antibiotic, Methacin (methylolgramicidin) in a 1:10,000 concentration. This antibiotic, derived from Tyrocidin does not have the hemolytic properties of Tyrocidin. Vonecidin has self-sterilizing properties.

Davis has used vonecidin for the treatment of acute rhinitis, acute nasopharyngitis, acute pharyngitis and acute sinus infections.

He has noted no disturbance in olfaction such as has been reported from the use of tyrothricin, and the vonecidin solution when dropped or sprayed into the nose had no irritating effects. Vonecidin has not yet been released by the manufacturer for general clinical use.

GROVE.

PHARYNX

The Recurrence Rate in Adenoids.

Lundgren, N.: Acta Oto-laryngologica 37:50 (Feb.) 1949.

In a large series of children examined after adenoidectomy, recurrence was noted only in 12%. Operations had been performed carefully with direct inspection of the nasopharynx. When symptoms of obstruction persist after operation the possibility of allergy should be kept in mind.

HILL.

Behcet's Syndrome.

Faaborg-Andersen, K.: Acta Oto-laryngologica 36:146, 1948.

Behcet's syndrome is characterized by recurring blebs in the mucous membranes of the mouth, pharynx and larynx, together with ulcerations in the genitals and uveitis. Its etiology is unknown, although it has been ascribed to a virus. There seems to be no effective treatment. A case is reported.

HILL.

The Question of Local or General Anaesthesia in Tonsillectomy, with Special Reference to the Frequency of Certain Complications.

Key-Aberg, Hans: Acta Oto-laryngologica 36:51, 1948.

Three thousand cases of tonsillectomy, 90% under local anesthesia, are reported with 1.9% bleeding after operation. The author compares this with 2.8% in Kettel's 500 cases. He reports no lung abscess in this series and prefers local anesthesia to general.

PRATT.

A Case of Primary Nasopharynx Tuberculosis?

Wille, Camillo: Acta Oto-laryngologica 36:61-65, 1948.

A case of presumptive nasopharyngeal tuberculosis is reported in a 17-year-old boy seen because of epistaxis. The source of bleeding was ascribed to a small tumor in the nasopharynx, which was removed. Histopathological examination was reported as mucous polyp with specific tubercular inflammation. No other evidence of tuberculosis could be found.

PRATT.

Evaluation of the Effects of Radiation on Non-malignant Lesions of the Nasopharynx.

Cutler, M. H., Marcus, Richard E., and Lederer, Francis: Radiology 52:816 (June) 1949.

A series of 50 cases (adults and children) with lymphoid hyperplasia about the eustachian tube orifice were divided into four groups. Each patient received irradiation according to the method advocated and later modified by Crowe. They were observed over the period of one year.

Group I consisted of 13 cases of nerve or conductive deafness. No improvement was noted audiometrically.

Group II consisted of 25 cases, children from four to nine years, except two young adults. The outstanding feature was a tendency to allergy or an allergic family background. All had had tonsils and adenoids removed. Considerable improvement in nasal and eustachian tube ventilation resulted.

Group III comprised 8 patients with symptoms suggestive of eustachian tube obstruction. Four of the patients noted that some of their symptoms were improved.

Group IV comprised 3 cases of chronic aural discharge. There resulted no change in character or amount of discharge.

JORSTAD.

LARYNX

Researches about Ossification and Conformation of the Thyroid Cartilage in Men.

Roncallo, P.: Acta Oto-laryngologica 36:110, 1948.

Fifty-seven larynges obtained postmortem were studied by means of roentgenograms. The author finds that ossification begins at

about 20 years of age, starting in the lower posterior zone. In men of advanced age, this may become complete. Rarely does this occur in women. He feels that calcification and ossification are two distinct processes and may be differentiated both histologically and radiologically. At times there is an anomaly of the upper horns, characterized by a deviation of the left horn towards the midline.

HILL.

BRONCHI

Mediastinal Emphysema Occurring During an Acute Paroxysm of Bronchial Asthma.

Grossman, J. W., and Cramer, O. S.: Radiology 52:705 (May) 1949.

This is a report of mediastinal emphysema in a 19-year-old white female occurring during an acute paroxysm of bronchial asthma. The patient felt something "snap" in her upper chest, and following this her neck began to swell. Dyspnea and difficulty in swallowing solid foods were the chief symptoms.

A chest radiograph showed mediastinal collection of air and subcutaneous emphysema of the neck. The patient was treated for her asthma and in two weeks the mediastinal emphysema had cleared.

Twenty-eight cases have been reported in the literature (through 1946). All patients recovered under conservative treatment.

JORSTAD.

Chronic Bronchitis. Never a Complete Diagnosis.

Lister, W. A.: Lancet 1:719-721 (Apr. 30) 1949.

Lister draws attention to the fact that "chronic bronchitis" is usually not a diagnosis but is a catch-basin for a variety of conditions, and in his experience the great majority of the patients exhibiting the symptomatology of cough, shortness of breath and the production of sputum are in fact sufferers from a mild degree of asthma. One of the states which is widely misdiagnosed as chronic bronchitis, especially in elderly people, is a mild degree of left ventricular failure.

He believes that the condition labeled "chronic suppurative bronchitis" is usually due to a focus of sepsis in the upper respiratory tract and that an infected antrum is often the cause. If a focus of infection cannot be found in the sinuses the chest should

be x-rayed for evidence of some local disease of the lung such as bronchiectasis, cystic disease, abscess, or possibly cancer. Chronic senile tuberculosis may drag on for years under the label of chronic bronchitis. Bronchitis is often due to chronic irritation from tobacco. Silicosis must be excluded.

In all the conditions enumerated above some degree of bronchospasm may mask the primary condition. Having enumerated various chest conditions which are frequently called chronic bronchitis there remain numerous other conditions for which no more accurate diagnosis than "chronic bronchitis" can be made. Regarding these, Lister would like to make three points:

1. That asthma, i.e., an allergic bronchial spasm, is the essential element of all this group.

2. That generalized emphysema is the end-result and a hallmark of an underlying asthmatic state.

3. That infection may occasionally be a primary cause, but is far more commonly secondary.

GROVE.

Broncho-pulmonary Segments of the Lung and Their Terminology.

Hardie-Neil, J., and Gilmour, W.: Brit. M. J. 2:309-310 (Aug. 6) 1949.

"The term 'broncho-pulmonary segment' is applied to a branch of the stem of a lobe of a lung which has on it secondary lobules. A broncho-pulmonary segment with its secondary lobules is surrounded by connective tissue derived from the visceral pleura." From pathological data it is apparent that infection in such a segment may pass to the periphery or pleura but only gross pathology such as tuberculosis or malignancy can break through the segmental boundaries to invade the neighboring segments.

This is an anatomical paper from the otolaryngological and pathological departments of the Aukland, New Zealand, hospital and must be read in its entirety by those interested.

GROVE.

ESOPHAGUS

Obstruction of the Gastro-oesophageal Junction.

Allison, P. R.: Lancet 2:91-94 (July 16) 1949.

This article covers a study of 507 cases of obstruction at the lower end of the esophagus seen during the last ten years in the

thoracic surgical department of the General Infirmary, Leeds, England. Three hundred and forty cases were malignant and 167 cases were nonmalignant. Of the 340 malignant cases, 184 were squamous carcinomata of the esophagus and 156 were gastric adenocarcinomata.

Of the 167 nonmalignant cases one was a leiomyoma, 60 were cases of cardiospasm and 106 were cases of simple ulcer. Cardiospasm occurred mostly in the age group 30-40 years and was twice as common in females as in males. Peptic ulcer occurred mainly in the age group 50-70 years and was as common in males as in females. Malignant obstruction of the cardia occurred mostly in the age group 50-60 years and was twice as common in males as in females.

In the rest of the article the author reviews the symptomatology of and the usually accepted treatment for obstruction of the cardiac end of the esophagus.

GROVE.

Treatment of Perforations of the Oesophagus.

Temple, Leslie J.: Brit. M. J. 1:935 (May 28) 1949.

Contrasting the former black prognosis for perforation of the thoracic esophagus most of which are caused by perforating foreign bodies or instrumentation, Temple calles attention to the fact that some of these cases can be saved by early primary suture. He publishes two case reports, with recovery in one after early primary suture. He asserts that a more conservative attitude can be taken for suspected perforation of the cervical esophagus on the ground that this region is under direct observation, and he reports a case where such conservative treatment with antibiotics was carried out with recovery. He states, however, that at the first sign of infection or extravasation outside the esophageal wall the surgeon must be prepared to explore the esophagus and suture the tear.

GROVE.

MISCELLANEOUS

Sequelae of Meningococcal Meningitis in Children.

Matthews, J. D.: Lancet 2:149-150 (July 23) 1949.

Matthews reports a survey of 50 children with meningococcal meningitis, 43 of which were aged two or less. Seven, or 14% died. The children were all treated with one of the sulfonamides with or without the addition of antimeningococcal serum and penicillin.

Only four of the survivors had sequelae. One child, aged 13 months, had hydrocephalus, hemiplegia, optic atrophy and was mentally defective and deaf. The other three children had deafness as the only complication.

Matthews believes that adequate chemotherapy reduces the case mortality in the very young as well as in the older age groups and he points out that the incidence of serious sequelae in this age was very small.

GROVE.

Leucocyte Counts in the Prevention of Drug Agranulocytosis.

Young, C. J.: Brit. M. J. 2:261-263 (July 30) 1949.

From a brief review of the literature it would seem to Young that both experimental and clinical evidence indicate that in drug agranulocytosis the condition is inaugurated in the bone marrow sometime before granulopenia develops in the peripheral blood and that the information given by the latter is too late for prophylactic purposes.

No evidence has been found that serial leucocyte counts during the administration of agranulocytosis-producing drugs either prevent agranulocytosis or reduce case mortality. Reliance on leucocyte counts is undesirable as it gives rise to a misguided sense of security which may prevent the adoption of more useful measures. Where prolonged sulfanilamide administration is necessary, the development of a sore throat or fever should be an indication to stop medication. Probably the best method of prophylaxis is to limit the period of sulfanilamide administration to a week or ten days.

GROVE.

Case of Salmonella Enteritidis Septicaemia with Lung Abscess.

Baird, I. McLean, and Capper, L.: Brit. M. J. 2:316-317 (Aug. 6) 1949.

Salmonella enteritidis is an acute gastro-intestinal illness with pyrexia and having a striking similarity to typhoid fever. The recorded complications include meningitis, pyelitis, osteomyelitis, cholecystitis, cholangitis and pulmonary involvements. The authors report a case which came to autopsy. During the illness the patient complained of sharp pain in the chest. At necropsy the whole right upper lobe of the lung consisted of a sloughing pneumonitis with central cavitation and there was a thin-walled abscess in the left upper lobe.

According to the authors only two previous cases of pulmonary involvement could be found in the literature. This rarity of pulmonary involvement is in striking contrast to the comparative frequency of this complication in typhoid and paratyphoid fevers and in infection with Salmonella choleraesuis.

GROVE.

Eosinophilic Xanthomatous Granuloma with Honeycomb Lungs.

Parkinson, Thomas: Brit. M. J. 1:1029 (June 11) 1949.

The precise nature of the Hand-Schüller-Christian syndrome is unknown. It is probably a phase in a generalized disorder of the reticulo-endothelial system and it has certain features in common with eosinophilic granuloma of bone.

Parkinson reports the case of a man, aged 56, who had diabetes insipidus, a cystic femur and a reticular mottling of the lungs on x-ray examination. Tomography confirmed the presence of small, cystic areas in the lungs. This case exhibited bone, pituitary and lung lesions consisting of diffuse infiltration and polycystic or honeycomb lungs.

More recently it has become apparent that the local bony granuloma may be part of a generalized disorder affecting the skeleton and the viscera and that other generalized reticuloses, namely, the Hand-Schüller-Christian disease and the acute fatal reticulosis of infancy, called the Letterer-Siwe disease, may give rise to bony changes identical to those of eosinophilic granuloma. Therefore, these are probably all variants of a common pathological disorder. The term "eosinophilic xanthomatous granuloma" has been offered as descriptive of the main histological features.

That pulmonary infiltration occurs in eosinophilic xanthomatous granuloma has been recognized since 1928. The disease occurs in acute and chronic form. In the complete variety there is pituitary, bone and pulmonary disease with possibly other visceral manifestations. In the incomplete forms, which are not uncommon, pulmonary, pituitary and bony lesions may occur singly or in any combination.

In the lung, fibrosis and cyst formation take place and at times this may be the sole manifestation of eosinophilic xanthomatous granuloma.

Irradiation may relieve the pain of the bone lesion but has no effect upon the pituitary or pulmonary manifestations of the disease.

GROVE.

Acute Pneumonitis in a Beryllium Worker.

Riddell-Royston, G.: Brit. M. J. 1:1030 (June 11) 1949.

Riddell-Royston describes the case of a man of 30 years who had worked in the laboratory of a radio factory for five weeks when he developed dyspnea on exertion and a slight nonproductive cough. He was admitted to a hospital as a suspected case of tuberculosis. Sputum was negative for tubercle bacilli. X-ray examination of his chest showed a soft mottling in some areas which were almost millet seed in size. This was distributed all over the lungs. The picture remained unchanged for six weeks and began to show resolution, and in another month complete resolution had taken place with no evidence of fibrosis.

Riddell-Royston calls attention to a report by Van Orstrand et al. in 1945 which described 128 cases of respiratory manifestations over a period of four years. Thirty-eight of these, including five fatalities, were described as chemical pneumonitis. X-ray changes were usually not present until the second or third week of the disease and cleared in from one to four months. Treatment was symptomatic. Penicillin and the sulfonamides were useless.

In 1947 Hardy and Tabershaw reported 17 cases of delayed chemical pneumonia. Four of them came on while the patients were still exposed to beryllium after periods of exposure varying from eight months to five years. Two of them died.

GROVE.

The Experimental and Clinical Use of Antihistaminic Drugs.

Hunter, R. B.: Edinburgh M. J. 56:54-61 (Feb.) 1949.

Histamine is a normal constituent of living tissue but held there in an inactive form and only produces its profound pharmacological effects if released as free histamine. When intravenously injected in man it is a vasodilator. When injected into animals it causes a spasm of plain muscle. Such spasm does not occur in a healthy human subject but in the asthmatic it produces a bronchospasm. These asthmatics have an idiosyncracy for the lung. Antihistaminic drugs will relieve this histamine-produced bronchospasm but will have little effect in relieving the naturally occurring bronchospasm.

After the introduction of several very toxic antihistaminic drugs, antergen was produced by Halpern in 1942. It was nontoxic enough to permit clinical use. Neoantergen was introduced

by Bovet in 1944 and is marketed as anthisan. Benadryl and pyri-
benzamine appeared in 1945.

Acording to Halpern the antihistaminic drugs act to block the
tissue receptors for histamine, thereby preventing the histamine from
producing its customary effects. These drugs have two other prop-
erties, (1) a local anesthetic effect and (2) and anti-acetylcholine
effect. Both anthisan and benadryl have atropine-like properties.

Antihistaminic drugs have an action on nervous tissue result-
ing in a diminution of the flare (not the wheal) when histamine is
injected into the skin.

Neither histamine nor acetylcholine produces skin itching or
burning independently but in combination they do. The antihista-
mine drugs allay this itching and burning because they upset the
histamine-acetylcholine partnership.

The dosage and therapeutic application of the antihistaminic
drugs are covered and their use in various dermatological conditions
such as urticaria, angioneurotic edema and pruritis is assessed.

In hay fever the use of antihistaminic drugs is so encouraging
that they are displacing the desensitization treatment in the milder
cases. In severe cases Hunter advises a course of desensitization fol-
lowed by the oral administration of the antihistaminics during the
season. He warns against increasing the dosage of the antigen under
cover of antihistaminic therapy to avoid severe reactions.

In treating perennial rhinitis the results of administering these
drugs were not as good as in hay fever and while in hay fever 75%
of patients had symptomatic relief of symptoms this was only true
in 51% of the cases of perennial rhinitis. However, more than one-
half of these patients were free of symptoms three months after treat-
ment ceased.

After controlled experiments in administering antihistaminic
drugs to asthmatic patients Hunter concludes that they have not
proven of value in the treatment of asthma.

The side effects of drowsiness, fatigue, nausea and dryness of
the mouth are not dangerous and can be minimized by giving the
drugs after a meal.

GROVE.

A Case of Hand-Schüller-Christian Disease.

Jokipii, S. G.: Ann. Med. Int. Fenniae 37:220, 1949.

A patient with complete deafness was diagnosed as having
Hand-Schüller-Christian disease on the findings of bone defects in

the skull, pulmonary changes resembling miliary tuberculosis, diabetes insipidus and disturbances in the development of the genitalia. The deafness was attributed to disorders of lipoid metabolism although there was a past history of suppurative otitis media. One might question the diagnosis upon the reported findings.

HILL.

On Abscess of the Lung.

Anfforine, V. M., and Laitinen, H.: Ann. Med. Int. Fenniae 37:183, 1949.

In a discussion of 106 cases of lung abscess the authors found penicillin-sensitive aerobic cocci the usual causative agent and felt that combined sulfonamide-penicillin therapy gave the best results. Surgical intervention could be postponed safely for a longer period than two months. Prognosis for those cases due to *Bacillus coli* was frequently unfavorable. Embolic abscesses are usually single while bronchiectasis is prone to cause multiple abscesses. Blood sedimentation rate is of help in indicating the progress of the disease.

HILL.

Notices

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AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations in 1950:

January 8-11 in New Orleans, Louisiana, at the Hotel St. Charles.

May 17-20 in San Francisco, California, at the Hotel Mark Hopkins.

October 3-6 in Chicago, Illinois, at the Palmer House.

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THE NATIONAL COMMITTEE FOR CHILE

The National Committee for Chile is now receiving gifts for the library of the medical school of the University of Chile at its new collection center in the Library of Congress, Washington. The newer materials in the library, including periodicals, books and reference materials, were totally destroyed in the recent fire. Medical periodicals of the last ten years and recent medical books are urgently needed. Your contribution will be appreciated.

National Committee for Chile, Room 318, Library of
Congress, Washington, D. C.

II PAN-AMERICAN CONGRESS OF OTORHINOLARYN-GOLOGY AND BRONCHO-ESOPHAGOLOGY

The II Pan-American Congress will be held at Montevideo and Buenos Aires January 8-15, 1950.

The official themes will be as follows:

1. Frontal sinus.

2. Tumors of the pharynx.

3. Lesions of the mouth, pharynx and larynx caused by fungi or parasites.

4. Methods of treatment of laryngeal cancer to preserve function.

5. Bronchial obstruction in children.

6. Noncancerous lesions of the esophagus.

7. Speech perception.

8. Hearing aids.

9. Otosclerosis.

Presentations will be confined to these subjects. The official languages will be Spanish, Portugese and English.

Secretary: Dr. Pedro Regules, Calle San José 1092, Montevideo, Uruguay.

NOTICE

The ANNALS will purchase back numbers at $1.00 each, of the following which are out of print:

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